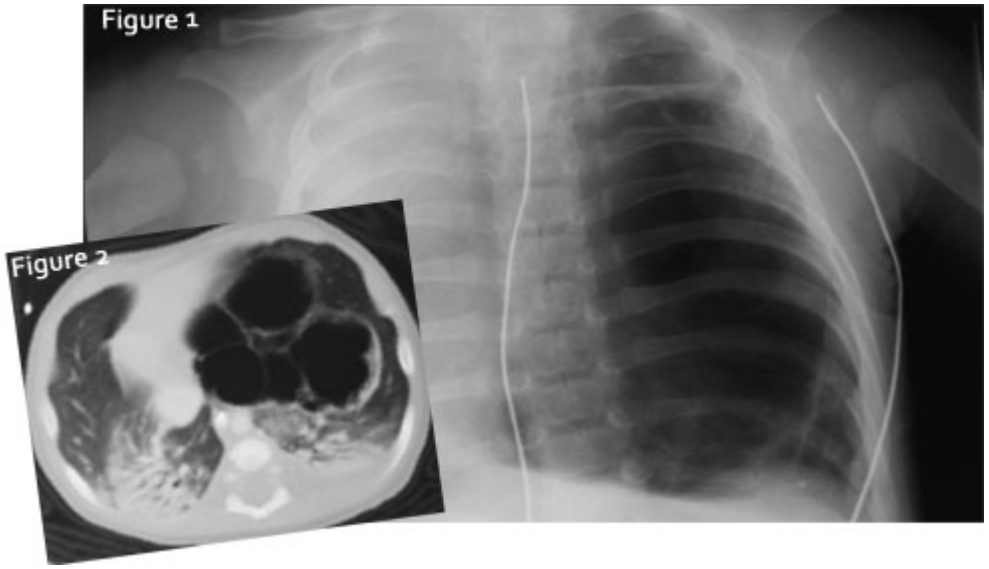
OSCE 5

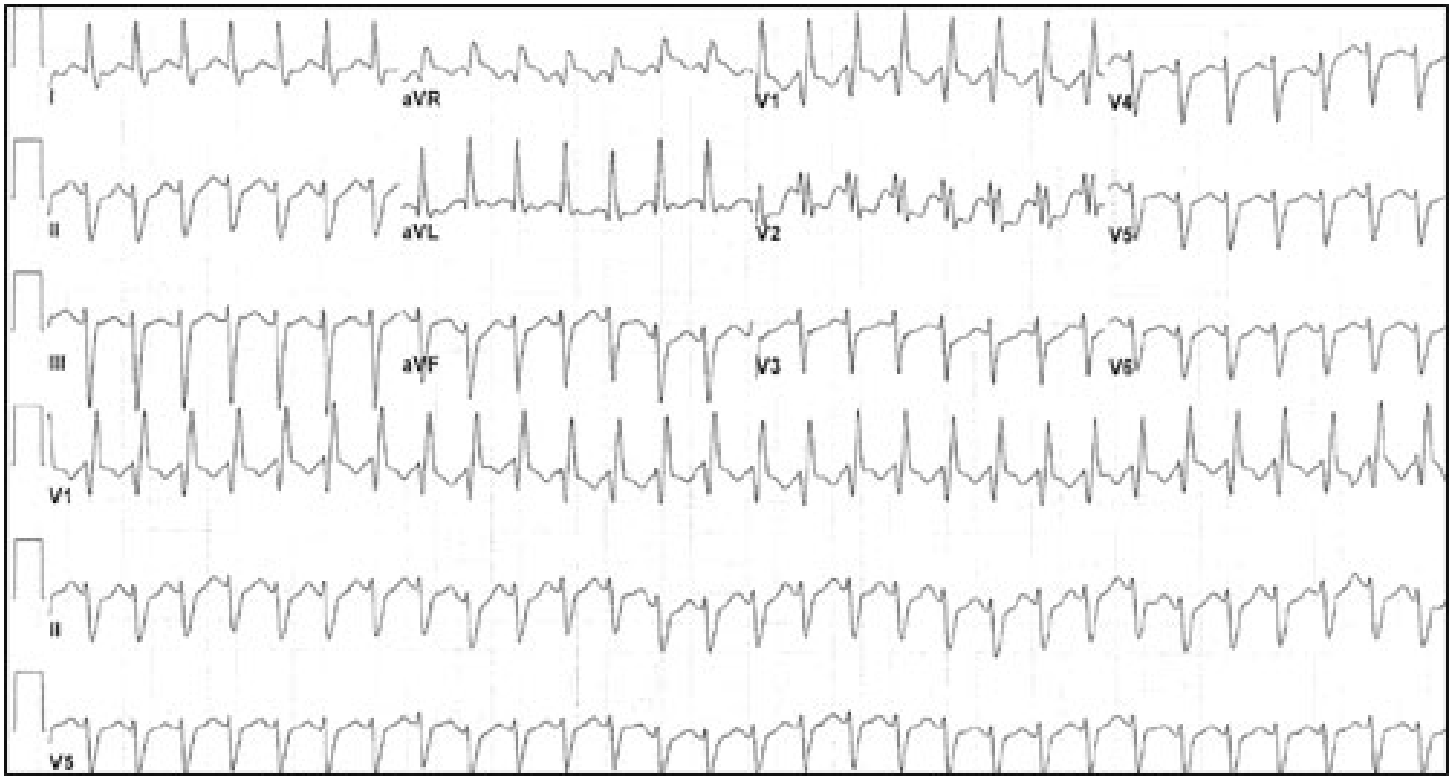
A 2-month-old Hispanic boy was brought to the emergency department because of a 1-day history of cough, nasal congestion, a few episodes of nonbilious vomiting, and poor oral intake



WHAT'S YOUR DIAGNOSIS?

Diaphragmatic hernia
Intrapulmonary sequestration
Congenital cystic adenomatoid malformation
Congenital lobar emphysema

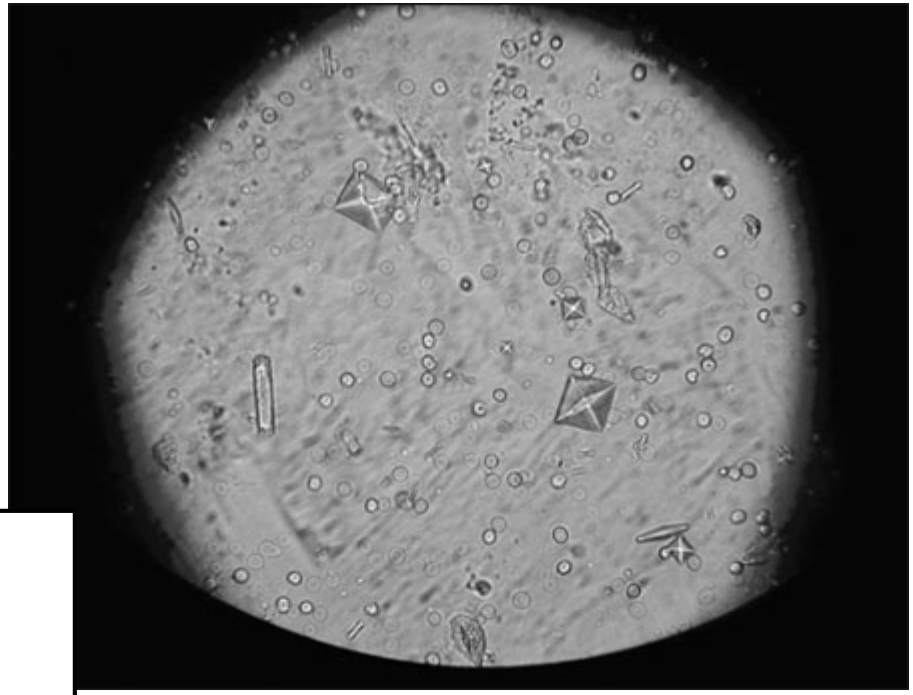
A 60-year-old man presented to the emergency department with palpitations that had started several hours earlier



- **Diagnosis: Supraventricular tachycardia (SVT) with right bundle branch block (RBBB).**
- In our patient's ECG, an RS complex is present in the precordial leads, the RS complex has a duration of less than 80 ms, and there is no atrioventricular (AV) dissociation. Because these three findings are not sufficient to diagnose or rule out ventricular tachycardia (VT), the V_1 and V_6 morphology are important diagnostically. The presence of rSR' in lead V_1 favors a diagnosis of SVT over VT. Although the R/S ratio in V_6 is less than 1, which would indicate VT, a diagnosis of SVT was made because both leads do not demonstrate criteria for VT.
- Access to a baseline ECG in a patient with bundle branch block (BBB) is another way to distinguish between SVT and VT. The baseline ECG in our patient, which was obtained during a routine physical examination several years earlier, showed a similar QRS complex, confirming the diagnosis of SVT (Figure 2).The small notch immediately following the QRS complex in V_1 is indicative of a possible P wave. Based on this finding, the differential diagnosis included atrial tachycardia, AV nodal re-entrant tachycardia, and AV re-entrant tachycardia.
- Wide QRS complex has been defined as a QRS duration of more than 120 ms. In our patient's ECG, this is almost 128 ms. The differential diagnosis of wide-QRS-complex tachycardia includes (1) VT, (2) SVT with underlying BBB or tachycardia-related aberrancy, (3) pre-excited SVT with anterograde conduction across an accessory pathway, or (4) an artifact.

- Brugada criteria can help clinicians differentiate between VT and SVT with reasonable probability.[2] These criteria include the following four findings:
- No RS complex in the precordial leads;
- Longest interval in any precordial lead from the beginning of the R wave to the deepest part of the S wave when an RS complex is present and exceeds 100 ms;
- Presence of atrioventricular dissociation; and
- QRS complex in leads V_1 and V_6 fulfilling certain morphological criteria for VT.

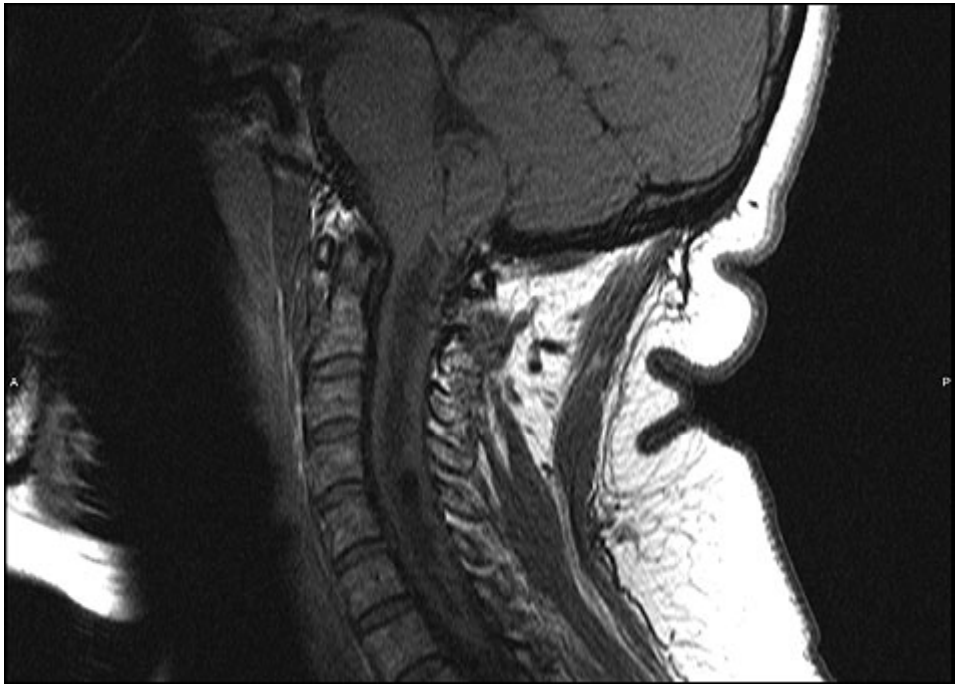
A 34-year-old man was brought to the hospital after his family found him unresponsive. Aside from an altered sensorium, his physical examination was normal. Laboratory studies showed severe metabolic acidosis and urinary crystals (Figure).



What is the diagnostic approach and what is the treatment?

- Ethylene glycol was detected in a serum assay, and the patient was treated with fomepizole (Antizol) and hemodialysis. His condition rapidly improved. On follow-up 3 weeks later, he was asymptomatic, and he admitted to swallowing ethylene glycol in a suicide attempt

A 58-year-old black woman with an 8-year medical history significant for seizure disorder and chronic headaches presented to the hospital with heaviness, swelling, tingling, and numbness of her right shoulder, all of which radiated to her right upper extremity



What's Your Diagnosis?

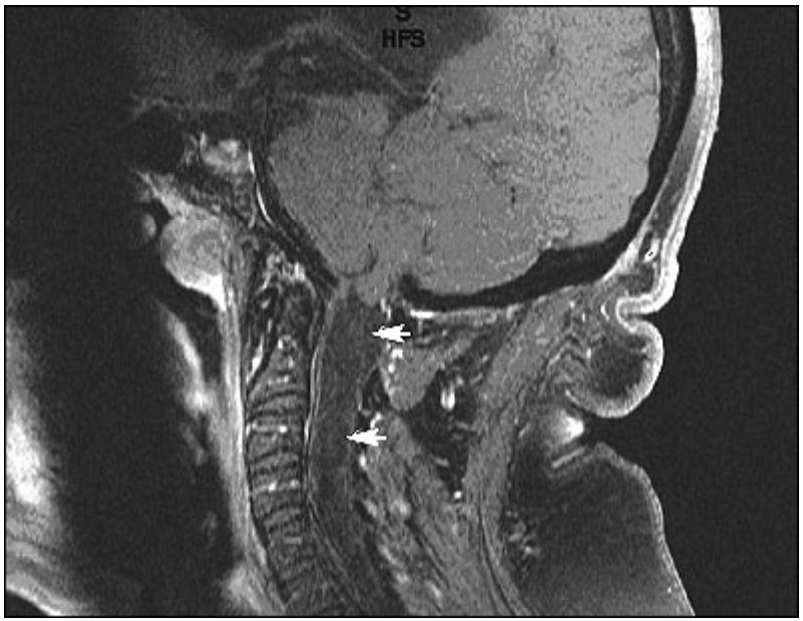
Polyneuropathy

Cervical disk prolapse

Multiple sclerosis

Syringomyelia due to Chiari I malformation

- **Syringomyelia due to Chiari I malformation**—The magnetic resonance image (MRI) shows a cervical syrinx that extends to the upper thoracic spinal cord and a tonsillar protuberance approximately 7 mm beyond the foramen magnum (Figure). Based on these findings, a diagnosis of syringomyelia in association with Chiari I malformation was made.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A 52-year-old woman presented to the emergency department reporting a 1-week history of rash on her bilateral lower extremities and "spots under her tongue." She noted no similar rashes in the past nor any recent trauma, viral infection, fever, sick contacts, overt bruising, hematuria, melena, hematochezia, epistaxis, gingival bleeding, or menorrhagia. Her medical history included lupus, congestive heart failure, hypertension, hypothyroidism, and anemia of chronic diseasePhysical examination revealed petechiae over her bilateral lower extremities as well as palatal and subungual petechiae (Figures 1, 2). The lesions were asymptomatic and the remainder of her physical examination was normal. Laboratory studies revealed a white blood cell count of 4,000/µL (normal, 4,500-11,000/µL); hemoglobin of 10.7 g/dL (normal, 12.0-15.0 g/dL), which was a stable level compared with her baseline; hematocrit of 35.9% (normal, 35%-45%); and a platelet count of 6 $x10^3$/µL (normal, 14-40 $x10^3$/µL). Her liver function tests, Epstein-Barr virus titer, human immunodeficiency virus test, and hepatitis panel were all negative.

What's Your Diagnosis?

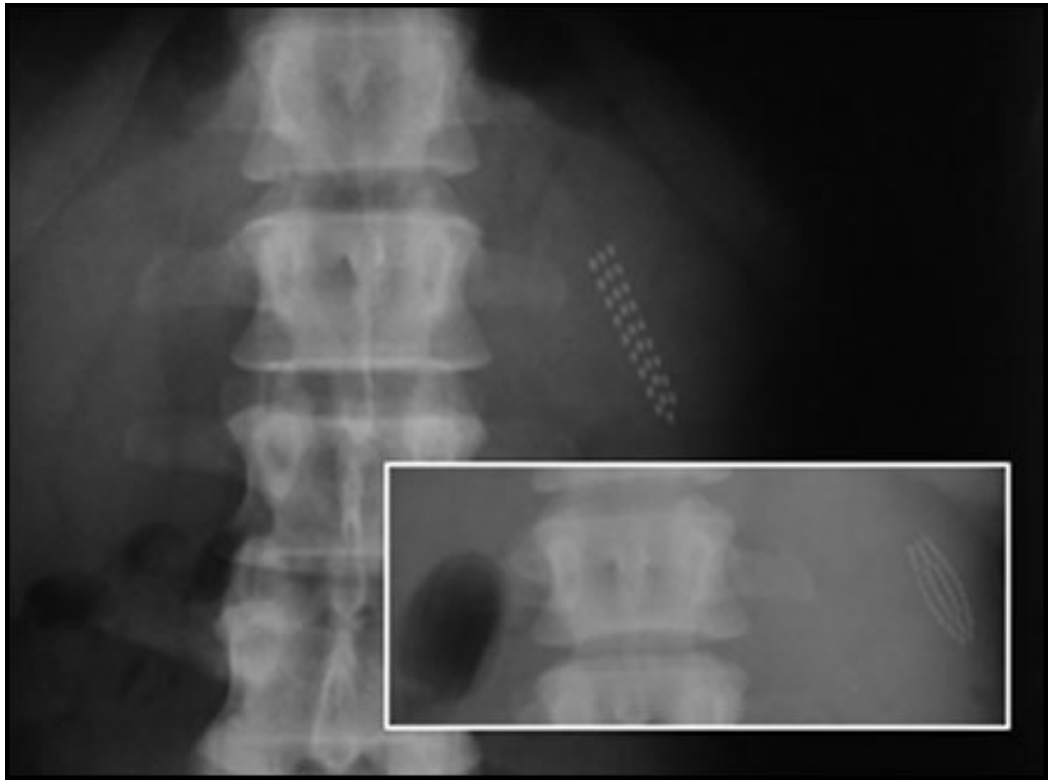
Chronic disseminated intravascular coagulation

Idiopathic thrombocytopenic purpura

Leukocytoclastic vasculitis

- **Idiopathic thrombocytopenic purpura (ITP)**—The patient was admitted to the hospital, after which a diagnosis of ITP was made. She was treated with prednisone, 100 mg daily, and a 5-day course of intravenous immunoglobulin (IVIG). During hospitalization, her platelet count reached a high of 27 $x10^3/\mu L$, and she experienced no overt bleeding.

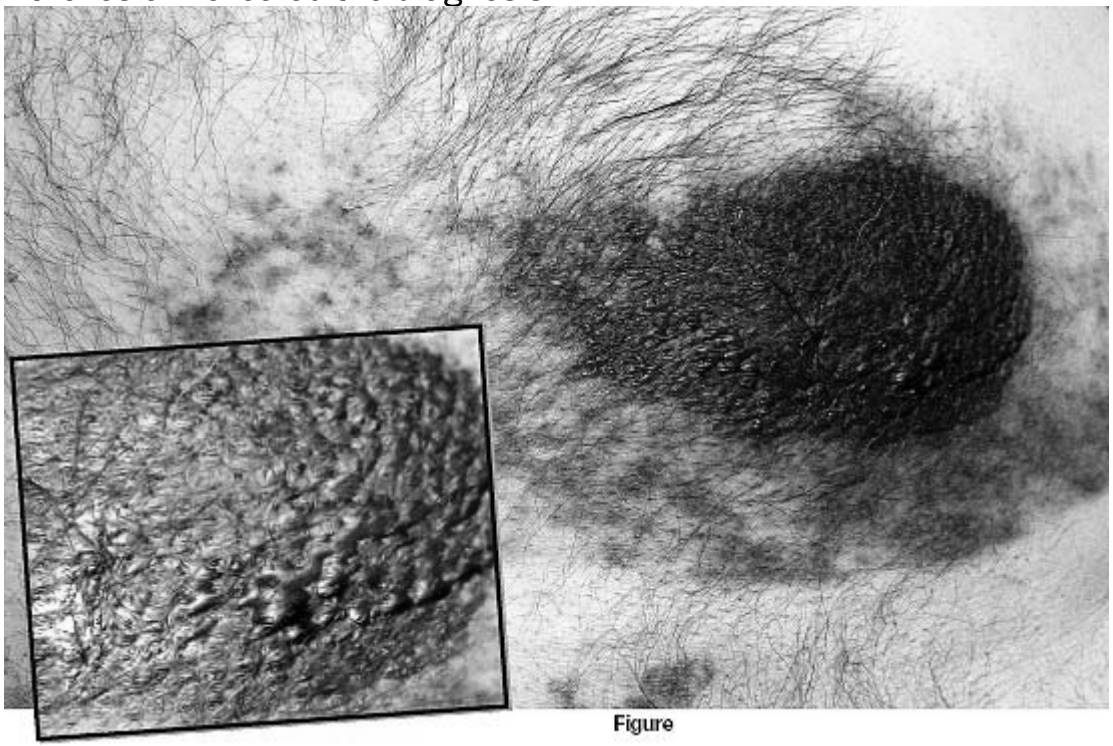
A 28-year-old man presented with abdominal pain, nausea, and a single episode of vomiting. Significant comorbities included depression and bipolar disorder. Physical examination was unimpressive except for multiple laparotomy scars.



What is your diagnosis?

- A plain radiograph of his abdomen showed a foreign body in the left upper quadrant, resembling a toothbrush (Figure). A careful retrospective history from the patient and family revealed that the patient had swallowed a toothbrush on a previous occasion. The patient underwent an uneventful laparotomy to have the toothbrush removed

An 80-year-old man with diabetes, hypertension, chronic kidney disease, and coronary artery disease was admitted to the hospital for chest pain. segment elevation myocardial infarction. Other medications initiated in the hospital included metoprolol, simvastatin, insulin, and calcitriol for secondary hyperparathyroidism (parathyroid hormone level >150 pg/mL). At 12 hours after admission, a skin lesion was noted at the site of the insulin administration on the abdominal wall (Figure). A biopsy of the lesion revealed the diagnosis.



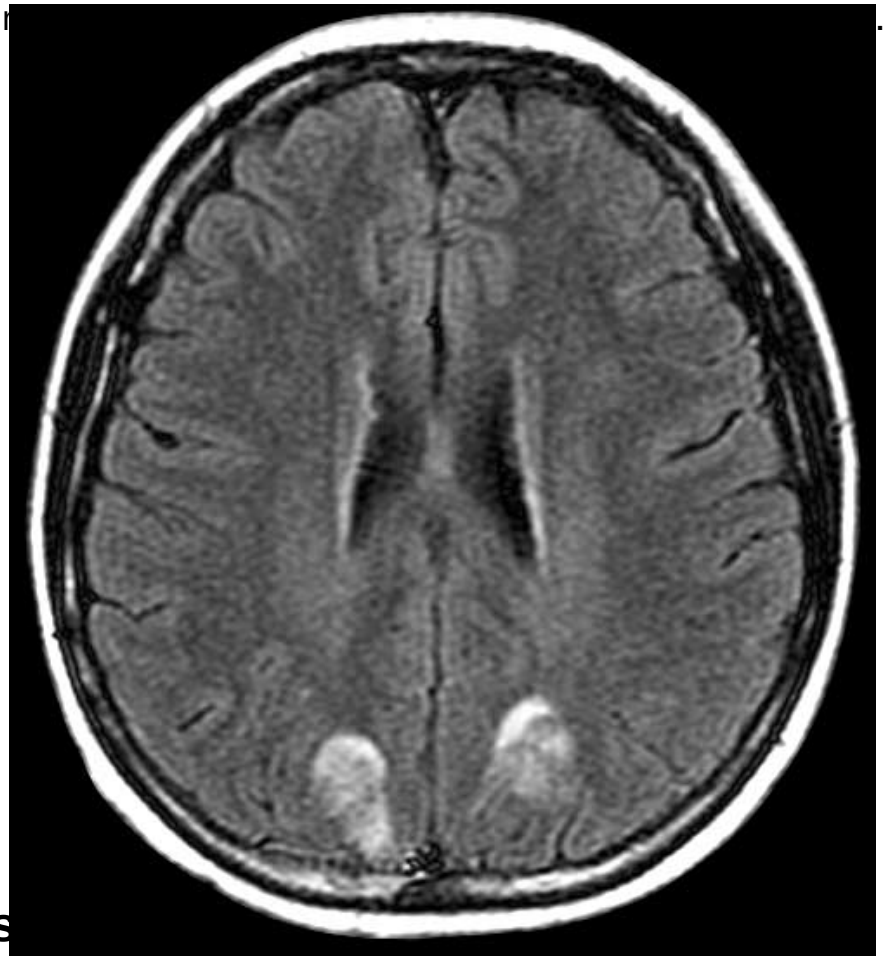
Figure

What's Your Diagnosis?

Calciphylaxis
Heparin-induced skin necrosis
Subcutaneous hemorrhage
Cholesterol embolization

- **Subcutaneous hemorrhage**—The skin biopsy confirmed the diagnosis. The likely precipitating event was subcutaneous insulin injection at that site, in addition to an elevated activated partial thromboplastin time (aPTT) from heparin infusion. Heparin is a known cause of subcutaneous and deep-tissue hematomas in patients with a prolonged aPTT. Unless the patient is hemodynamically unstable, the treatment is usually conservative. In this case, the heparin therapy was stopped, which contained the hemorrhage. The skin biopsy was negative for thrombi or necrosis (heparin-induced skin necrosis), medial calcification (calciphylaxis), and cholesterol clefts (cholesterol emboli).
- Calciphylaxis is mostly seen in patients with end-stage renal disease who are on hemodialysis or who have recently received a renal transplant. Calciphylaxis is related to vascular and soft-tissue calcification and can be precipitated acutely by the initiation of calcitriol therapy for secondary hyperthyroidism. The diagnosis is confirmed by skin biopsy, which shows arterial occlusion and calcification in the absence of vasculitic change.
- Heparin-induced skin necrosis is a rare entity that has been described only in patients receiving subcutaneous heparin.

A 21-year-old woman was brought to the emergency department after having 2 episodes of generalized tonic-clonic seizures. The patient had a complex medical history that included systemic lupus erythematosus (SLE), hemodialysis-dependent status after bilateral nephrectomy, thrombotic thrombocytopenic purpura (TTP), and poorly controlled hypertension. A computed tomography (CT) scan of the brain (performed at an outside hospital) was reportedly normal. Magnetic resonance imagi



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What's Your Diagnosis

Lupus cerebritis

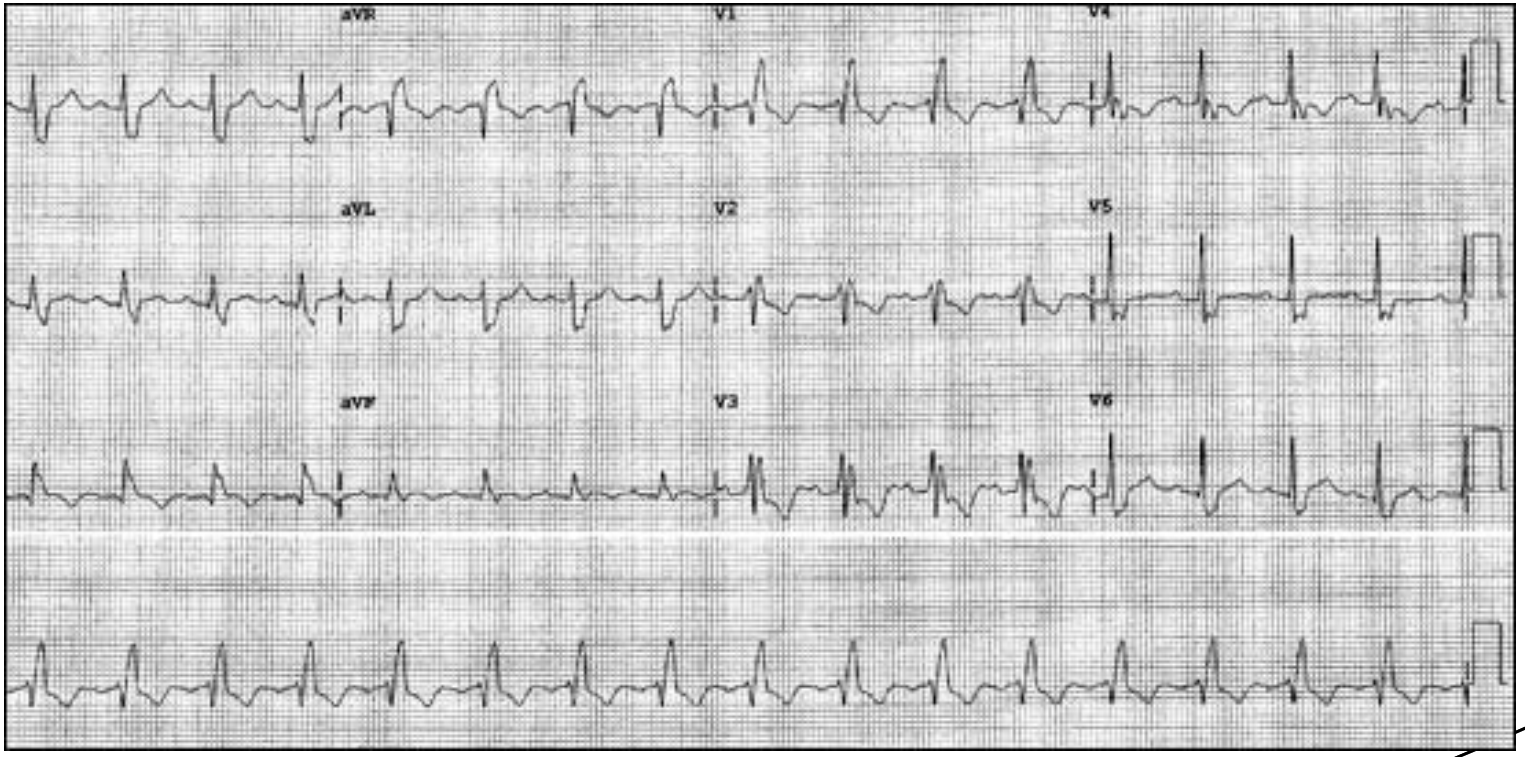
Acute infarction

Posterior reversible encephalopathy syndrome

Acute disseminated encephalomyelitis

- **Posterior reversible encephalopathy syndrome**—MRI findings included regions of high signal on T2 and fluid attenuation inversion recovery (FLAIR) sequences within the posterior parietal and occipital lobes. These sequences indicated edema in the posterior subcortical white matter, as well as cortical involvement. The patient was admitted to the hospital for blood pressure (BP) control. Repeat MRI 10 days later demonstrated a normal brain, with complete resolution of the previous T2/FLAIR signal abnormalities (Figure 2). This reversibility of the lesion confirmed the diagnosis. The patient improved clinically and did not experience another seizure.

- A 51-year-old African-American man presents to the emergency department with a 1-week history of sudden onset of exertion-induced breathlessness associated with light-headedness. He has no fever, cough, chest pain, orthopnea, or paroxysmal nocturnal dyspnea. His history includes type 2 diabetes and hypertension. His vital signs are: blood pressure, 157/100 mm Hg; heart rate, 93 beats/min; respiratory rate, 15 breaths/min; temperature, 98.7° F. The physical examination is remarkable for pallor and bilateral pedal edema, with no evidence of jugular venous distention or tenderness in the lower limbs. Lungs are clear to auscultation. Arterial blood gases on room air are: pH, 7.48 (normal range, 7.35-7.45); PCO_2, 29.0 mm Hg (normal, 35-45); PO_2, 53.0 mm Hg (normal, 80-100); bicarbonate, normal (at 21.4 mmol/L); troponin, normal (at 0.15 µg/L); B-type natriuretic peptide, 217 pg/mL (normal, <100). A 12-lead electrocardiogram (ECG) is obtained


aVR
V1
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V2
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V3
V6

- **Diagnosis: Acute cor pulmonale caused by pulmonary embolism ($S_1Q_3T_3$ pattern).**
- Pulmonary embolism ranges from incidental, clinically unimportant thromboembolism to massive embolism with sudden death. Acute pulmonary embolism causes elevated pulmonary artery and right heart pressures and decreased cardiac output, which will be reflected in the ECG by the $S_1Q_3T_3$ pattern. However, when the ECG changes persist even after the pulmonary artery pressure and the right heart size have returned to normal, additional mechanisms are involved,[1] including the release of catecholamine or histamine at the cellular level.[2,3]
- This patient's ECG shows: sinus tachycardia (with a rate of 100/min), S wave in lead I,Q wave in lead III, and T-wave inversion in lead III ($S_1Q_3T_3$ pattern); Q waves are also seen in leads II and aVF; T-wave inversion is seen in leads V_1 through V_4 (anterior leads); complete right bundle-branch block (RBBB); and ST-segment depression in leads V_4 through V_6. These findings make this ECG very suspicious for pulmonary embolism. A computed tomography scan of the chest with contrast showed large bilateral pulmonary embolisms

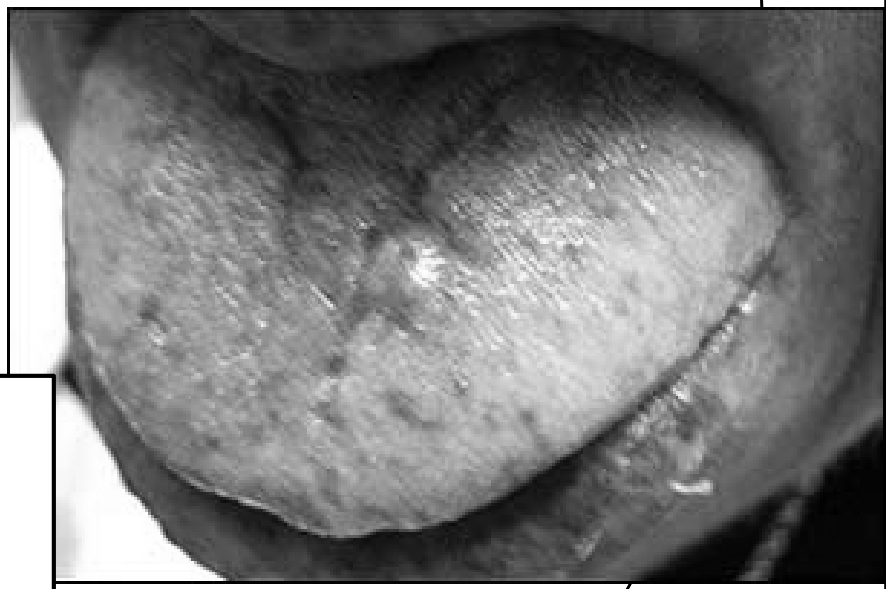
- A 59-year-old African-American woman with a history of renal failure and deep-vein thrombosis (DVT) was admitted to the hospital for altered mental status and confusion. At the time of admission, she was taking warfarin therapy for her DVT. During a magnetic resonance imaging (MRI) of her head, she had a generalized tonic-clonic seizure, which was not seen on the MRI.
- The patient was started on phenytoin (Dilantin) for seizure control. The following morning, her hand was edematous, with dark discoloration of the dorsal and palmar aspects (Figures 1, 2). She had no tenderness at the site, and there was no breakdown of the skin with gentle stroking. Her neurologic examination was normal, as was her range of motion.

What's Your Diagnosis?

Warfarin-induced skin necrosis
Raynaud's disease
Staphylococcal scalded skin syndrome
Purple glove syndrome

- **Purple glove syndrome**—This syndrome is a serious consequence of intravenous (IV) phenytoin administration, which can cause distal-limb edema, discoloration, and pain and may eventually result in skin necrosis and limb ischemia. This process takes place at the site of the IV administration of phenytoin within 2 hours. The mechanism is thought to be secondary to the extravasation of the highly alkaline phenytoin solution. Treatment involves removing all IV lines from the affected extremity, elevation of the extremity, and avoidance of blood pressure checks in that extremity. If no pulse is found or progression to skin necrosis occurs, a surgical consultation should be obtained.
- Our patient's condition was managed conservatively, including monitoring pulses, skin care, and follow-up with the plastic surgery department for possible skin grafting. No skin grafting was needed. Eventually, however, the patient had autoamputation of her fingers.
- Fosphenytoin (Cerebyx) may be a beneficial alternative for reducing the incidence of these potential side effects; however, its cost is a major disadvantage and is the reason for its reduced use. Purple glove syndrome has been reported in 6% of patients receiving phenytoin.[1] Therefore, physicians should monitor IV access sites when administering phenytoin or should consider the use of fosphenytoin instead.

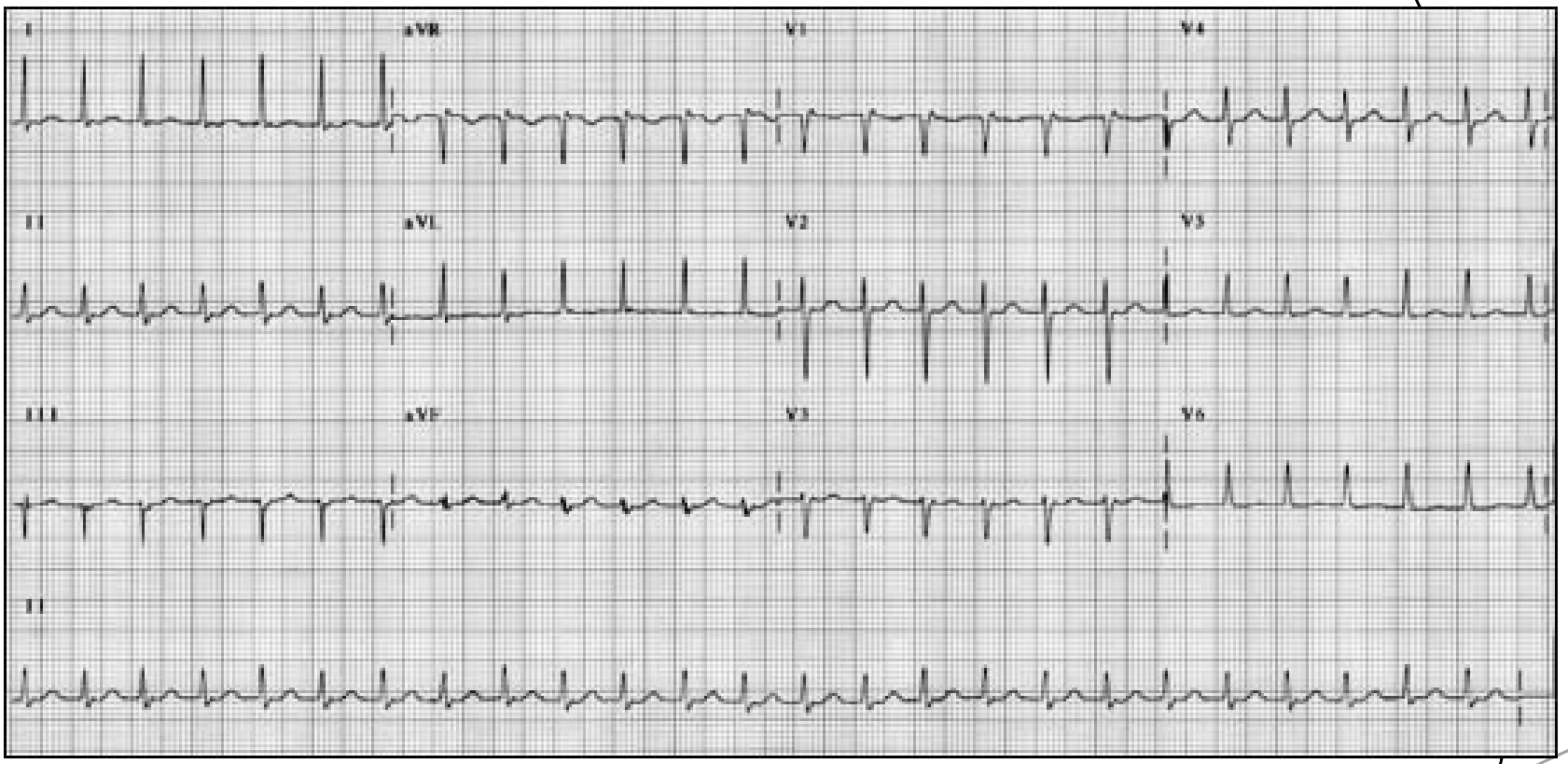
A 65-year-old woman with a 20-year history of recurrent epistaxes presented to the emergency department for her third episode of epistaxis in a month. Her mother also had a history of epistaxis. Physical examination showed telangiectasias on her fingers , lips, and tongue



What is your diagnosis?

- Osler-Weber-Rendu syndrome, also known as hereditary hemorrhagic telangiectasia, is an autosomal dominant disorder generally characterized by the triad of telangiectases, recurrent epistaxis, and a family history of the disorder. The major cause of morbidity and mortality is hemorrhage from these multiorgan arteriovenous malformations. Recurrent epistaxis is seen in up to 90% of patients. The most common physical findings are telangiectases of the skin, oral mucosa, nasal mucosa, and conjunctiva. Treatment is aimed at limiting the amount of hemorrhage and minimizing the sequela of chronic blood loss.

- An 89-year-old woman with a history of hypertension, type 2 DM, and a TIA 7 years earlier presented to the hospital with palpitations. She had no history of syncope. She has been taking enteric-coated aspirin 81 mg/day and metoprolol 75 mg twice daily. Physical examination findings included: afebrile; HR 150 beats/min; BP 116/76 mm Hg; and RR16 breaths/min; pulse oxygen saturation, 96%. Cardiac examination showed no evidence of jugular venous distension or carotid bruits; the first and second heart sounds were normal, with no evidence of murmurs, gallops, or rubs. Both lungs were clear to auscultation, and no peripheral edema was seen. At the time of the previous attack 7 years ago, her left ventricular ejection fraction was 70%. An electrocardiogram (ECG) was obtained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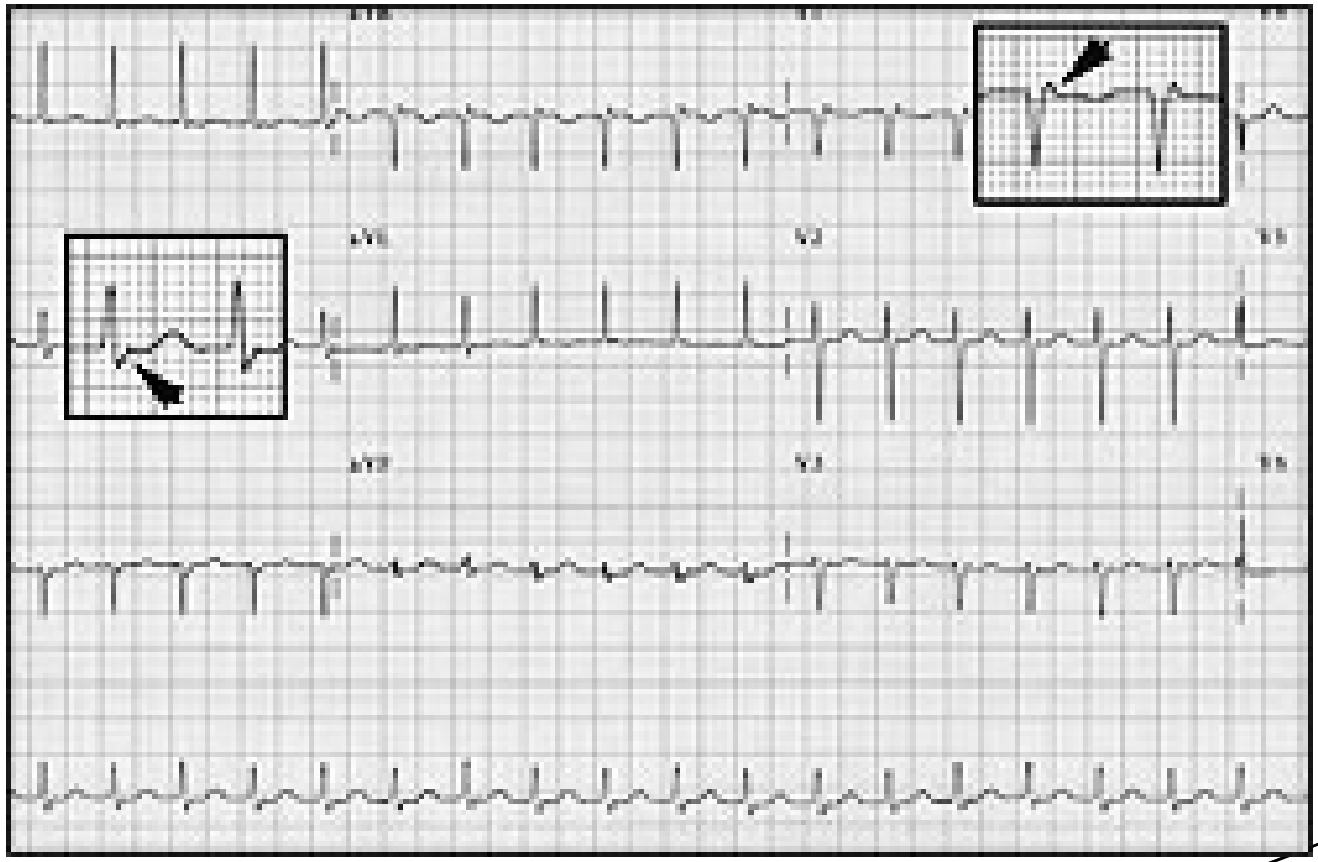
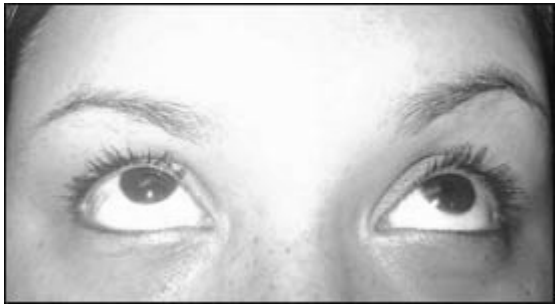
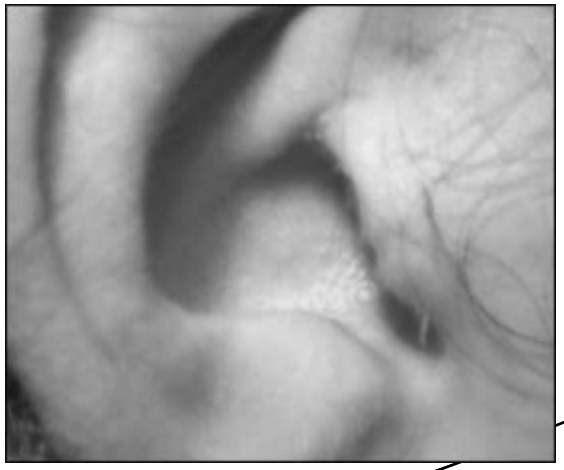


- **Diagnosis: Atrioventricular nodal reentrant tachycardia (AVNRT).**
- The 12-lead ECG at presentation (Figure 1) showed tachycardia with a regular rate of 150 beats/min and narrow QRS complexes. No P waves were seen on the ECG, but terminal S waves were seen in leads II, and aVF and terminal r' waves in V_1. The patient was given intravenous (IV) adenosine (Adenocard) 6 mg, which converted the rhythm to normal sinus, leading to a resolution of the S waves in leads II and aVF and terminal r' waves in V_1 (Figure 2). A diagnosis of AVNRT was made, because the terminal waves were found to be retrograde P waves, which are superimposed on the QRS complex and are known as "pseudo waves."

- A 27-year-old woman presented to our emergency department with a 1-day history of unilateral facial weakness and pain in the right ear. The symptoms started as right-ear pain and facial numbness around the periauricular area, then progressed to include loss of taste, finally manifesting as seen in Figures 1, 2, and 3.
- The patient also reported mild tinnitus in her right ear. She denied any trauma to her face, fever or chills, headache, body weakness, body rash, or blurry vision. She had no significant medical history and did not travel outside of the country recently. No masses were noted on the neck or head.



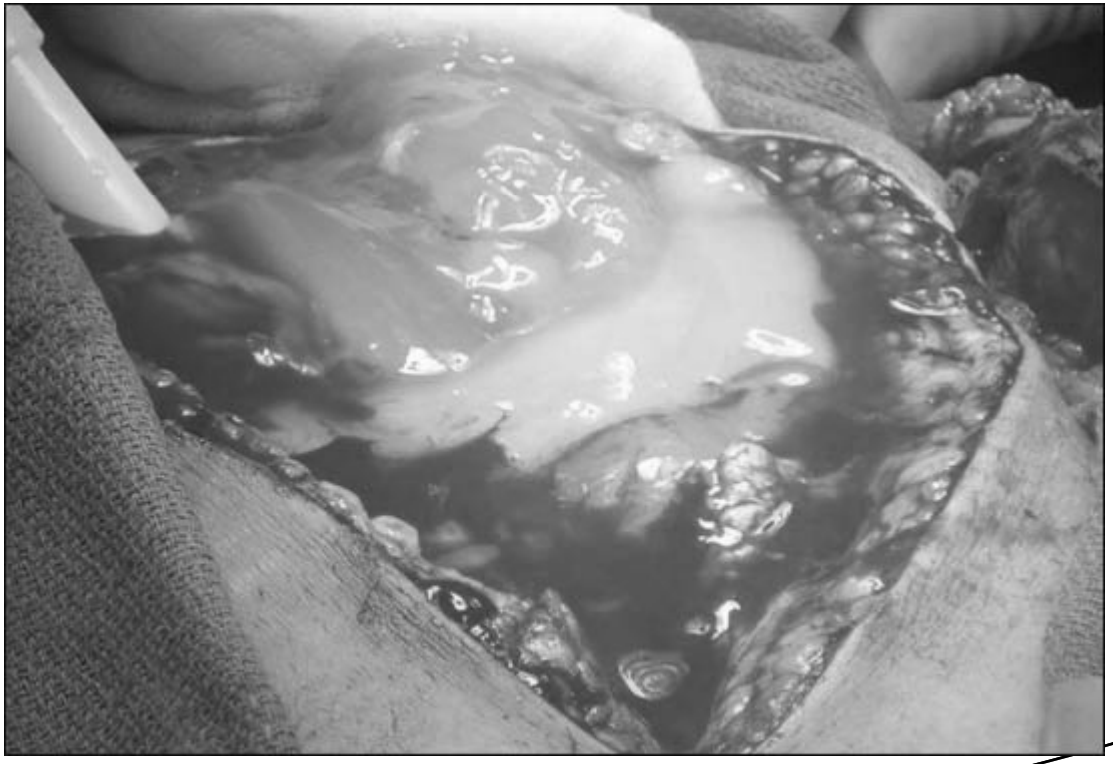


What's Your Diagnosis?

Acute otitis media
Ramsay Hunt syndrome
Lyme disease
Facial palsy from herpes simplex

- **Ramsay Hunt syndrome**—Also called herpes zoster oticus, Ramsay Hunt syndrome is the second most common cause of atraumatic acute peripheral facial nerve palsy in adults.[1] This syndrome represents a triad of symptoms: unilateral facial nerve palsy, ear pain, and eruption of vesicular herpetic lesions.[1,2] Unilateral dysfunction of the lower motor neuron of cranial nerve VII (peripheral facial palsy) is the common manifestation, which involves the upper and lower face (Figure 1), including the inability to raise the eyebrow (Figure 2) and an absence of nasolabial fold on the affected side. The finding of a vesicular rash on the concha of our patient's auricle (Figure 3) confirmed the diagnosis of Ramsay Hunt syndrome

A 38-year-old woman presented with a 6-day history of a painless umbilical discharge. On physical examination, she looked well but had conspicuous ascites and a jellylike substance oozing from an ulcerated umbilicus. At laparotomy, the same type of substance filled her peritoneal cavity (Figure) and bound together all of her abdominal and pelvic organs.



What is your diagnosis?

- These findings were diagnostic of pseudomyxoma peritonei. No tumor cells were evident in sections of the peritoneum or the umbilicus.

- **Points to remember:** pseudomyxoma peritonei—also known as "jelly belly"—is a rare disorder characterized by diffuse collections of gelatinous material in the abdomen and pelvis along with mucinous implants on the peritoneal surfaces. It is associated with mucin-producing neoplasms—benign and malignant—originating most often in the appendix or in the ovary. The chief clinical features are gelatinous ascites, indolent course, and striking disparity between the extent of disease and the patient's healthy appearance. Repetitive "debulking surgery" is the mainstay of treatment; the 5-year survival rate is about 40%.
- **Diagnosis:** Pseudomyxoma peritonei.
-

- An 81-year-old man was rushed to the emergency department by ambulance after a syncopal episode following a bowel movement. He complained of abdominal pain that radiated to the thorax and back and reported feeling ill for the past several days, fatigue, and nonlocalized abdominal and back pain. He did not have emesis, chest pain, dyspnea, or recent trauma. His history included ischemic cardiomyopathy, congestive heart failure, mitral valve regurgitation, arthritis, hemorrhoids, a prolapsed rectum, an old hiatal hernia repair, and coronary artery bypass graft surgery performed a few years ago. Vital signs were: blood pressure, 97/59 mm Hg; pulse, 80 beats/min; respiration, 16 breaths/min. On physical examination, the abdomen was distended and rigid, with flanks of blue discoloration

What's Your Diagnosis?

Pneumothorax

Ruptured esophagus

Diaphragmatic hernia with bowel strangulation

Pulmonary mass fever

- **Diaphragmatic hernia with bowel strangulation—** The x-ray and CT scout film showed a massive right-sided diaphragmatic hernia (Figure 1), with evidence of bowel eventration and free air under the right diaphragm (Figure 2). A nasogastric tube was placed. The patient remained alert, oriented, and calm, but breathing required increased effort, and he became pale. Continued deterioration was expected, and rapid sequence intubation was performed. Immediately after intubation his blood pressure dropped to a pulseless 35/19 mm Hg. Life support was started, but resuscitative efforts were unsuccessful.

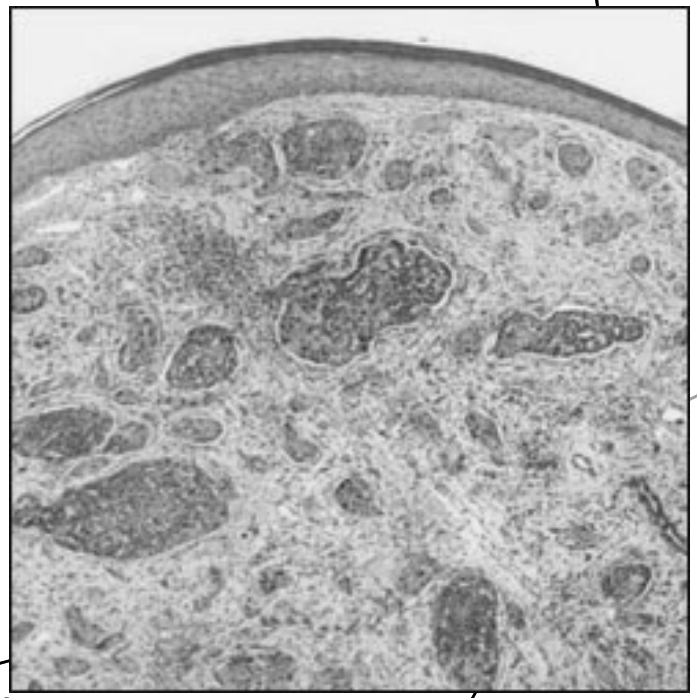
A 21-year-old college student presented with a 1 month's history of "wavy red bumps" underneath her skin. She had been seen by 2 other clinics before coming to our clinic. She had not responded to previous therapy with a topical antifungal agent and a course of cephalexin. The rash was mildly pruritic but not painful. Examination revealed many serpiginous vesicles and bullae on an erythematous base involving the right medial ankle (Figure 1), as well as the plantar and dorsal right foot, which had a large tattoo (Figure 2). She had no significant medical or family history

What's Your Diagnosis?

Allergic contact dermatitis
Cutaneous larva migrans
Granulomatous tattoo reaction
Impetigo

- **Quiz Answer**
- **Cutaneous larva migrans—**The patient had recently traveled to Belize and spent a considerable amount of time swimming and walking barefoot on the beach over several different days. The diagnosis was made based on this travel history and the clinical examination. Cutaneous biopsy is generally not required to diagnose cutaneous larva migrans.
- Cutaneous larva migrans is a self-limiting, serpiginous eruption caused by the penetration of animal (usually domestic dogs or cats) hookworms into the epidermis, after contact with soil contaminated with animal feces. The most common nematodes associated with this condition are *Ancylostoma caninum* and *Ancylostoma brasiliense*. In general, the larvae stay confined to the epidermis, because they do not produce collagenase, which is essential for penetration of the epidermal basement membrane. They tend to migrate 1 to 2 cm/day and most often affect the lower extremities, hands, and buttocks. Although cutaneous larva migrans is found worldwide, it is most prevalent in warm, moist, tropical clima

An 81-year-old white man presented with a rapidly enlarging nodule on his scalp. He had first noticed the lesion 3 weeks earlier. Since then, he had several episodes of bleeding, along with a purulent discharge, from the lesion. His non-Hodgkin's lymphoma was in clinical remission; his history also included Graves' disease and a recent diagnosis of pulmonary squamous-cell carcinoma. Physical examination confirmed the presence of a solitary, well-circumscribed, nontender, 1.3 x 1.3-cm erythematous nodule, with a friable center (Figure 1). No cervical lymphadenopathy was noted. A biopsy of the nodule was performed, and histologic analysis revealed the diagnosis (Figure 2).

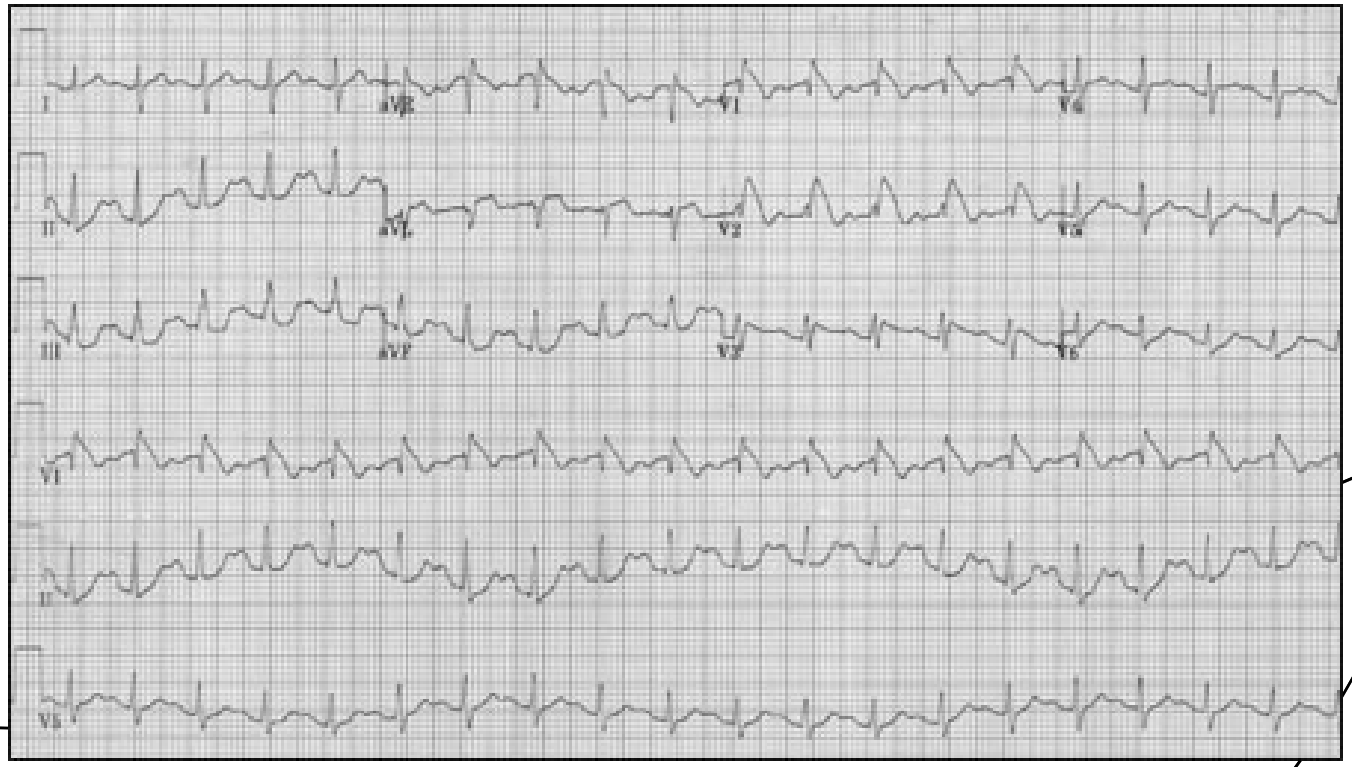


What's Your Diagnosis?

Basal-cell carcinoma
Sweet's Syndrome
Metastatic squamous-cell carcinoma
Keloid

- **Metastatic pulmonary squamous-cell carcinoma—**Computed tomography (CT) of the chest revealed numerous small masses in both lung lobes that had not been present on previous CT scans. The patient refused any further medical interventions. He developed progressive dyspnea and was placed in hospice care. He died 8 weeks later.

A 34-year-old white man of English descent presents with multiple episodes of diarrhea and SOB. He also complains of palpitations for the past 2 hours. The diarrhea, which began 6 hours earlier, is watery and nonbloody. He has had 1 to 2 loose bowel movements every hour for the past 6 hours. He complains of mild chest discomfort but denies nausea, vomiting, fever, or syncope. His travel and food histories are unremarkable, and he has not taken any antibiotics recently. His medical history is unremarkable, and he has no family history of syncope, sudden death, or sudden infant death syndrome. Physical examination shows: weight, 225 lb; blood pressure, 95/60 mm Hg; respiratory rate, 25 breaths/min; heart rate, 135 beats/min. The heart rhythm is regular, and no murmurs, rubs, or gallops are evident. Lung and abdominal examinations are normal, and no extremity edema is found



- **Diagnosis: Brugada syndrome.**
- The ECG shows the typical manifestations of Brugada type 1. In this patient, Brugada syndrome was unmasked incidentally when an ECG was obtained secondary to tachycardia and chest discomfort in the setting of a self-limited diarrheal illness. Brugada syndrome was later confirmed by electrophysiologic studies, in which ventricular tachycardia was induced, without the subsequent use of procainamide.
- This ECG shows ST-segment elevation in precordial leads V_1through V_3, with QRS morphology resembling a right bundle-branch block (BBB). The diagnosis of Brugada syndrome is based on clinical findings of syncopal or sudden death episodes in a patient with a structurally normal heart and a characteristic ECG pattern of right BBB and ST-segment elevation in leads V_1 through V_3.
- The clinical manifestations of the syndrome are caused by episodes of polymorphic ventricular tachycardia or ventricular fibrillation. When the episodes terminate spontaneously, the patient develops syncope. When the episodes are sustained, full-blown cardiac arrest and eventually sudden death occur. Thus, the manifestations of Brugada syndrome can range widely: at one end of the spectrum are asymptomatic individuals, at the other end are those who die suddenly. In addition, the ECG manifestations can be present intermittently, and therefore not all ECGs are diagnostic for this syndrome.

- A 68-year-old Caucasian woman with steroid-dependent rheumatoid arthritis and diverticulosis presented with progressive abdominal distension and pneumaturia of 1 week's duration. Plain abdominal radiograph revealed a massive radiolucent pelvic shadow (Figure), reported as a 25-cm, grossly dilated cecum suggestive of a cecal volvulus. While the patient was waiting for a computed tomography (CT) scan and possible emergency surgery, catheterization of the urethra produced a loud gush of air. Results of a subsequent CT scan of the abdomen and repeat abdominal radiograph were unremarkable.

What is your diagnosis?

- The presumptive diagnosis of colovesical fistula was confirmed intraoperatively, and the defect was repaired.
- Diverticulitis accounts for 40% to 90% of cases of colovesical fistula.
- **Points to remember:** Simple bedside urethral catheterization may help differentiate a dilated urinary bladder from a dilated sigmoid colon or cecum.
- **Diagnosis:** Colovesical fistula.
-

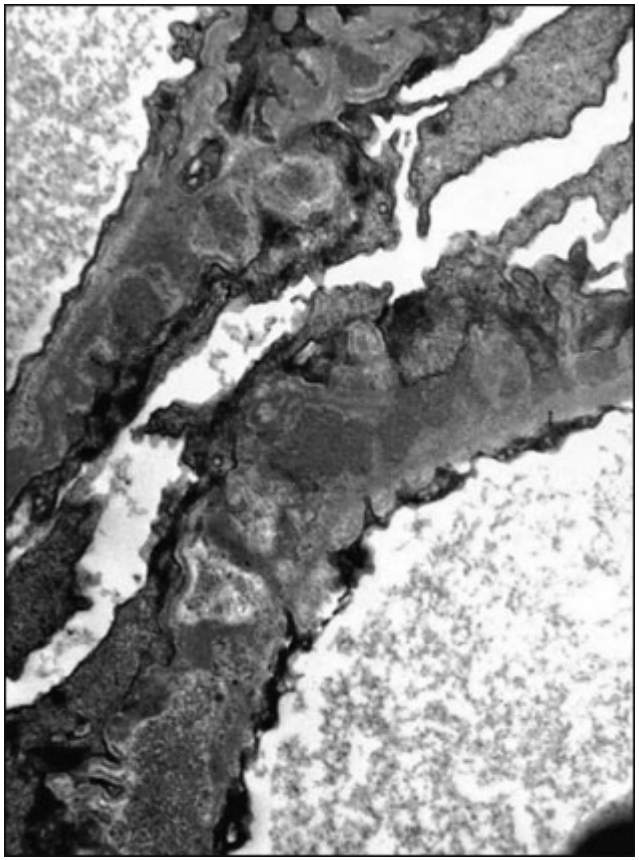
A 36-year-old man presented to the emergency department with diffuse maculopapular rash that began 4 days earlier. The lesions first appeared on the face and neck; 2 days later they progressed to the entire upper body, accompanied by fever (temperature, 101.1°F), headache, photophobia, and generalized aches and pains. Physical examination showed papules and macules predominantly on the face, neck, upper back (Figure 1), chest, upper extremities, and scrotal area. Axillary and inguinal lymph nodes were tender to palpation. Comprehensive test results were all within normal limits, as was the neurologic examination. ELISA testing for HIV antibodies and blood cultures were negative. Biopsy of the lesions revealed dermal lymphocytic infiltration (Figure 2).

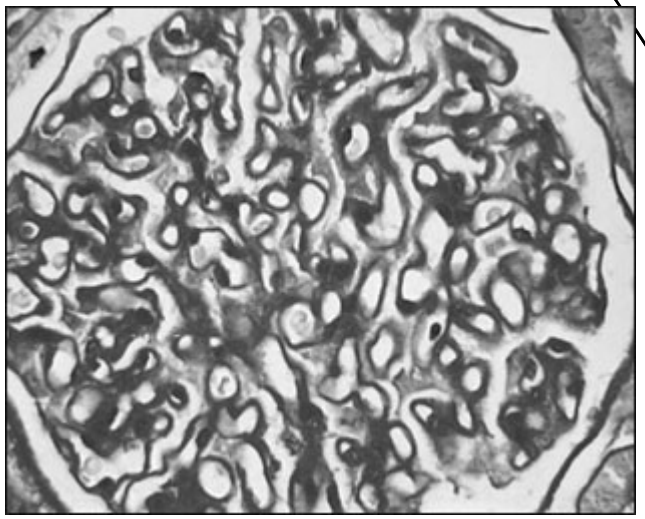
What's Your Diagnosis?

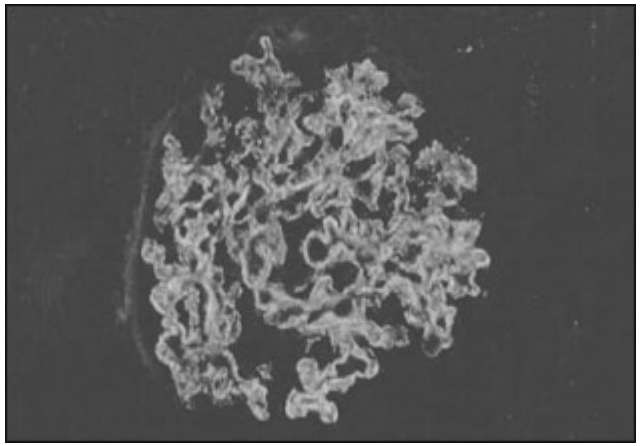
Primary Epstein-Barr virus infection
Syphilis
Acute retroviral (or HIV) syndrome
Rocky Mountain spotted fever

- **Acute retroviral (or HIV) syndrome—**The diagnosis of acute retroviral syndrome was made based on results of an HIV polymerase chain reaction test, which showed more than 500,000 copies/mL of plasma HIV-RNA.
- The initial manifestation of HIV infection in one half to two thirds of patients is a mononucleosis-like illness, referred to as acute retroviral syndrome, also known as acute HIV syndrome. The precise incidence of acute retroviral syndrome is not known but is reported to range from 40% to 90%.[1] The clinical features of this syndrome are nonspecific and variable; these include fever, lymphadenopathy, pharyngitis, and rash in about 70% to 90% of patients.[2] Additional presentations include headache, photophobia, and meningitis in about 12% of patients.[2] About two thirds of patients may have a truncal exanthem that may be maculopapular, roseolalike, or urticarial. Skin biopsies are nonspecific, revealing perivascular lymphocytic infiltrates and dermal mononuclear cell infiltrates

- A 61-year-old white man presents to our clinic complaining of anasarca, 15-lb weight gain over the past 4 weeks, excessive fatigue, and orthopnea of 1 month's duration. His medical history includes poorly controlled hypertension, hyperlipidemia, prostatectomy done for prostate cancer (with recent elevation of prostate-specific antigen), and thyroidectomy performed for hypothyroidism. He has been taking ibuprofen for repeat headaches. His regular medications include spironolactone, lactulose, irbesartan, bisoprolol/hydrochlorothiazide, levothyroxine, atorvastatin, and amlodipine. Physical examination shows: hypertension, ascites, and 3+ pitting edema of the lower extremities. Serum liver function testing reveals only severe hypoalbuminemia. Additional testing shows: 14.8 g of protein on 24-hour urine collection; creatinine clearance of 52 mL/min; and significantly elevated thyroid-stimulating hormone level (7.4 mIU/L). Results of a renal biopsy pointed out the diagnosis





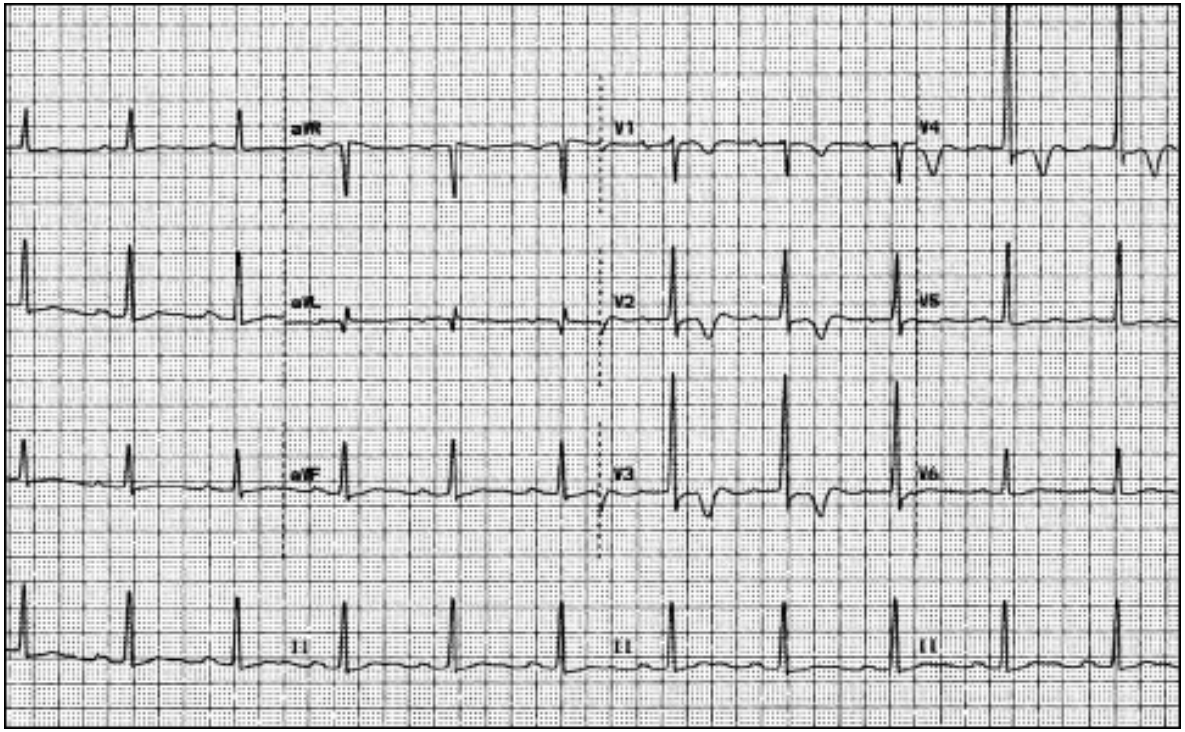


What's Your Diagnosis?

Focal-segmental glomerulosclerosis
Membranous nephropathy
Membranoproliferative glomerulonephritis
Minimal-change disease

- **Membranous nephropathy—**Results of the renal biopsy revealed the characteristic features of membranous nephropathy: light microscopy (Figure 1) showed diffuse thickening of the glomerular basement membrane, with normal cellularity. Immunofluorescence (Figure 2) revealed granular immunoglobulin G deposition and subepithelial deposits on electron microscopy (Figure 3).
- Membranous nephropathy and focal-segmental glomerulosclerosis are among the most common causes of the nephrotic syndrome in nondiabetic adults.[1] Membranous nephropathy is frequently idiopathic in adults but can be secondary to other conditions. Clinical, laboratory, and histologic features can distinguish between these 2 diagnoses. The causes of membranous nephropathy include rheumatoid arthritis; medications, such as gold and penicillamine; hepatitis B virus (HBV) and hepatitis C virus (HCV) infections; and systemic lupus erythematosus. Malignancy (primarily solid tumors) has been suggested as a cause in as many as 5% to 10% of cases in adults

An electrocardiogram (ECG) taken during a routine preoperative examination of a 79-year-old black woman who was scheduled for hemorrhoid surgery is shown (Figure 1). Physical examination (including blood pressure) was normal; no murmurs or abnormal heart sounds were found



What's Your Diagnosis?

Normal ECG variant

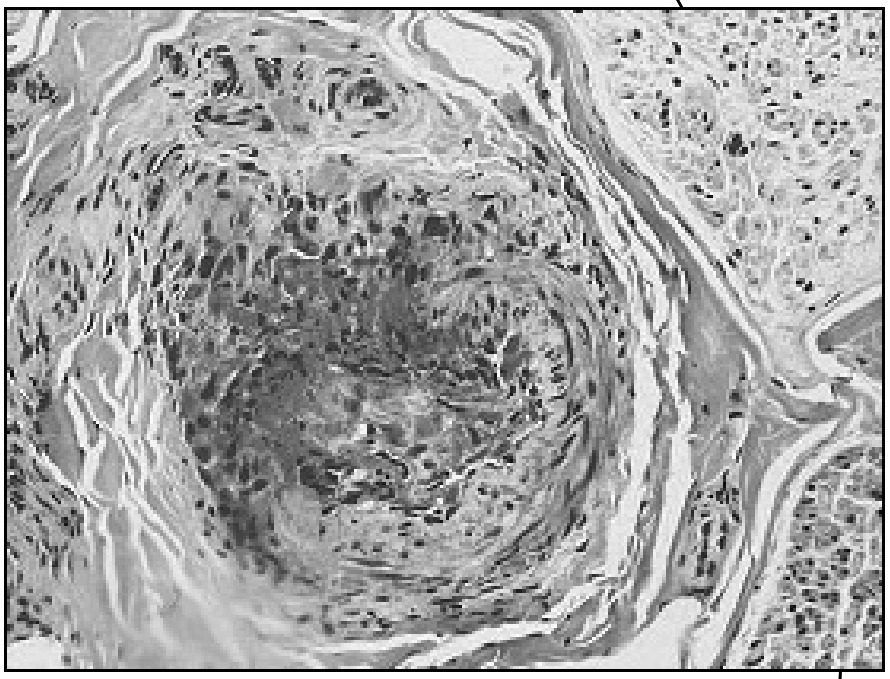
Apical hypertrophic cardiomyopathy

Aortic stenosis

Ischemic heart disease

- **Apical hypertrophic cardiomyopathy**—The ECG shows marked voltage of left ventricular (LV) hypertrophy, with deeply inverted T waves in leads V_2 through V_4, characteristic of apical hypertrophic cardiomyopathy. The echocardiogram confirmed the diagnosis of apical hypertrophic cardiomyopathy, demonstrating that the apical myocardium is markedly thickened

- A 36-year-old man presented with 2 weeks of progressive lower abdominal pain associated with nausea, vomiting, and 2 bloody bowel movements. Physical examination revealed a tender abdomen, with normoactive bowel sounds and erythematous plaques on the extensor surfaces of the elbows, knees (Figure 1), and metacarpophalangeal joints. Hyperreflexia was also evident. Upon his return from the first Gulf War, he was diagnosed with asthma. Skin plaque biopsy revealed a focal perivascular infiltrate of histiocytes and eosinophils, and a colonoscopy with random biopsies showed acute and chronic inflammation with eosinophilia (Figure 2). An echocardiogram revealed a left ventricular ejection fraction of 30%. Laboratory test results revealed 38% eosinophils and immunoglobulin E level of 844 IU. A diagnostic biopsy of the sural nerve demonstrated necrotizing vasculitis and eosinophilia involving the small muscular arteries

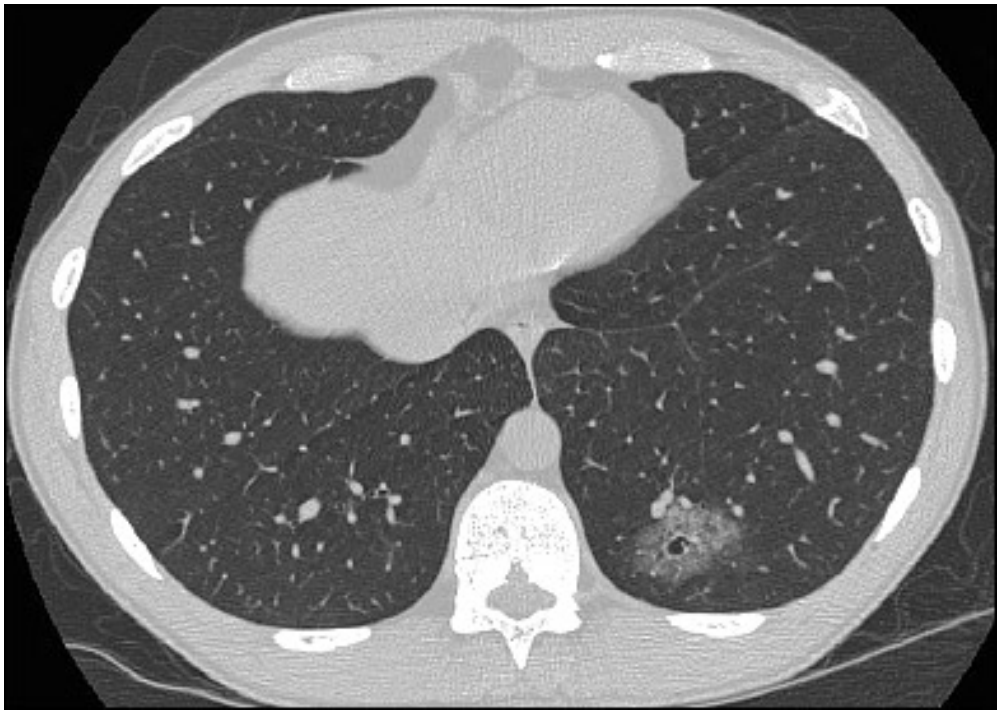


What's Your Diagnosis?

Wegener's granulomatosis
Polyarteritis nodosa
Churg-Strauss syndrome
Microscopic polyangiitis

- **Churg-Strauss syndrome**—This patient exhibits many of the typical features of Churg-Strauss syndrome. This condition may involve any organ, but involvement of the lungs, skin, cardiovascular system, kidneys, peripheral nervous system, or the gastrointestinal (GI) tract is common. Patients often present with nonspecific complaints of fever, malaise, anorexia, and weight loss characteristic of a multisystem disease.[1] Pulmonary findings usually dominate the clinical picture, with severe asthma attacks. Clinical signs of heart disease are common, as are cutaneous lesions; the latter include purpura and cutaneous and subcutaneous nodules. Peripheral neuropathy is seen in up to 75% of cases, often as a mononeuritis multiplex. Abdominal pain is seen in close to 60% of patients as an eosinophilic gastroenteritis

A 33-year-old emergency department resident physician had a positive tuberculin skin test 1 year after having a negative test. He had no clinical or radiologic evidence consistent with active tuberculosis (TB). Isoniazid was prescribed. Two months later he noted the onset of teaspoon-sized hemoptysis, which was repeated many times. His travel history included visits to Southeast Asia and South America. Physical examination was normal, as was a complete blood cell count. Three sputum smears and a culture were negative for *Mycobacterium*. The computed tomography (CT) scan of his chest is shown (Figure). All other tests were normal, including bronchoscopy and bronchoalveolar fluid

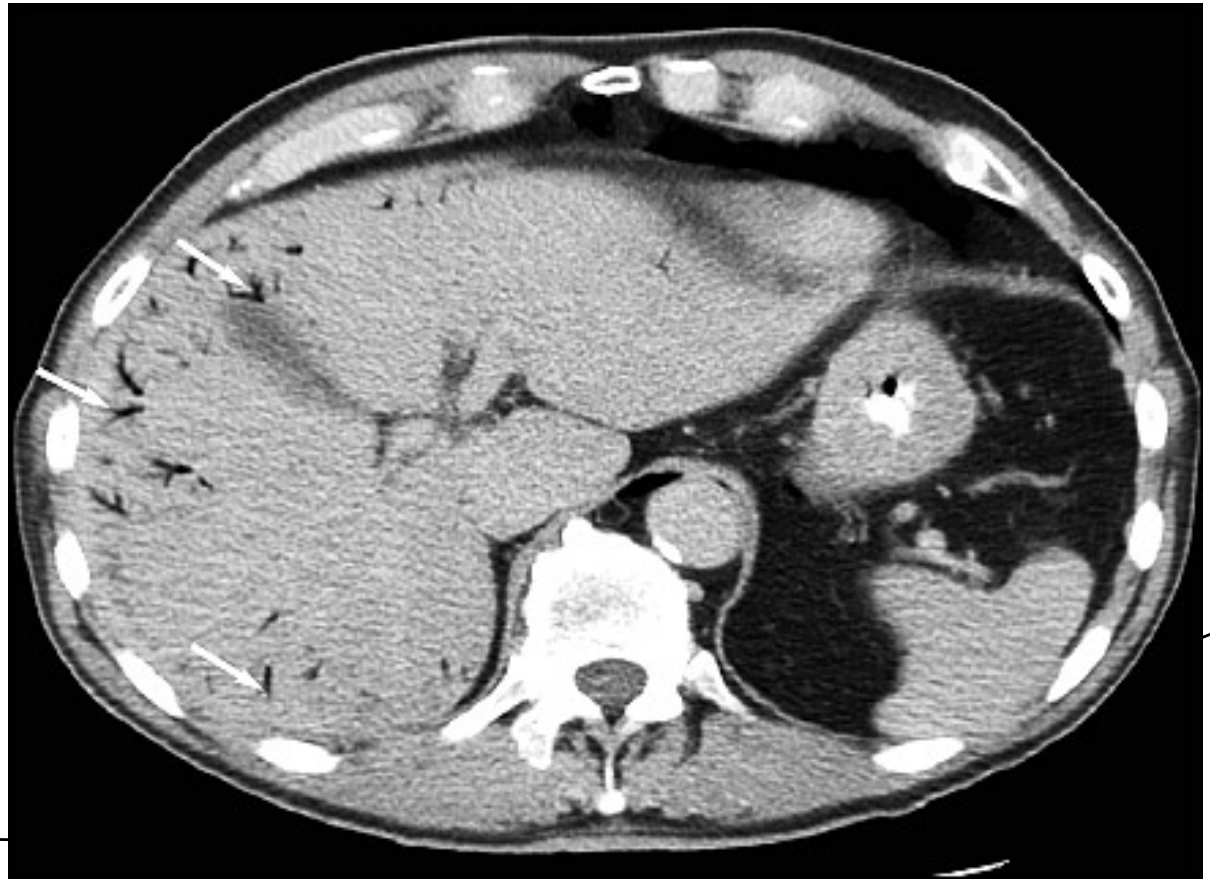


What's Your Diagnosis?

Tuberculosis
Aspergillosis
Paragonimiasis
Lung cancer

- **Paragonimiasis**—The chest CT revealed a 4-mm pulmonary cavity, with surrounding infiltrate in the left lower lobe (Figure). Human paragonimiasis is a subacute or chronic infection, usually of the lungs, caused by *Paragonimus* species. The patient's ELISA for immunoglobulin (Ig) G titer against *Paragonimus* was 1:64 (ie, positive

A 73-year-old man underwent esophagogastroduodenoscopy (EGD) with dilatation for an esophageal stricture. Shortly after the procedure, he developed neck pain, pleuritic chest pain, and dyspnea. Review of his history revealed no biliary tract manipulation. On physical examination, the patient was hemodynamically stable, with palpable crepitus in his neck anteriorly and mild epigastric tenderness without rebound or guarding. Computed tomography (CT) demonstrated extensive pneumomediastinum, pneumatosis of the gastric fundus, and hepatic venous air (Figure). Emergent esophagram revealed no extravasation of Gastrografin, suggesting no large perforation or tear. The patient received prophylactic antibiotics intravenously and was managed conservatively. Repeat CT revealed improvement, with no further extravasated air or abscess formation



- **Points to remember:** Complications of EGD can include bleeding, perforation, or esophageal tear. The finding of hepatic venous air in this patient can be explained by extravasation of air into the gastric veins, which return blood to the heart via the portal/hepatic venous system.
- **Diagnosis:** Hepatic venous air from gastroesophageal perforation.
-

A 78-year-old woman presented to the emergency department with chronic chest discomfort, shortness of breath, and fatigue. Her history included myocardial infarction (MI) 30 years earlier, heart failure, atrial fibrillation, and hypertension. A retired schoolteacher, she grew up in England and had immigrated to the United States more than 20 years ago. She denied any foreign travel since then or exposure to any construction trades, including mining.

Physical examination revealed a diffuse apical impulse, holosystolic murmur radiating to the axilla, and an S3 gallop.

What's Your Diagnosis?

Pericardial calcification

Calcified left ventricular (LV) aneurysm

Asbestosis

Cardiac echinococcosis

Calcified LV aneurysm—Figures 1 and 2 show marked calcification on the LV silhouette diagnostic of a calcified LV aneurysm, which occurs in a small percentage of patients with a large anteroseptal MI. LV aneurysm is a well-defined, thin, scarred or fibrotic wall (devoid of or containing necrotic muscle), resulting from a transmural MI that has healed over time. The calcified scar appears as a fine, curvilinear density, usually appearing on the anterolateral aspect of the heart or in the lower portion of the interventricular septum.

Pericardial calcification is associated with a history of pericarditis, most often related to tuberculosis but can also be linked to other etiologies, such as a viral process. Pericardial calcification can be ruled out in this case, because the calcification pattern seen on the imaging studies does not follow the contour of the pericardium. Unlike the calcification curving posteriorly, below the level of the pulmonary valve seen in Figure 2A, pericardial calcification extends along the pulmonary outflow tract

A 35-year-old Hispanic man with no significant medical history presented complaining of malaise, fever, and a nontender, enlarging right axillary mass of 2 months' duration (Figure). Laboratory test results were: platelet count, 54 x 109/L; hemoglobin, 99 g/L; and normal serum calcium and creatinine levels. Serum protein electrophoresis with immunofixation revealed elevations in gamma globulins (2.11 g/dL), immunoglobulin G (2150 mg/dL), and kappa chains (2110 mg/dL). A biopsy of the right axillary mass showed monoclonal plasma cells staining for clusters of differentiation (CD) markers CD138 and CD79A

What's Your Diagnosis?

B-cell lymphoma

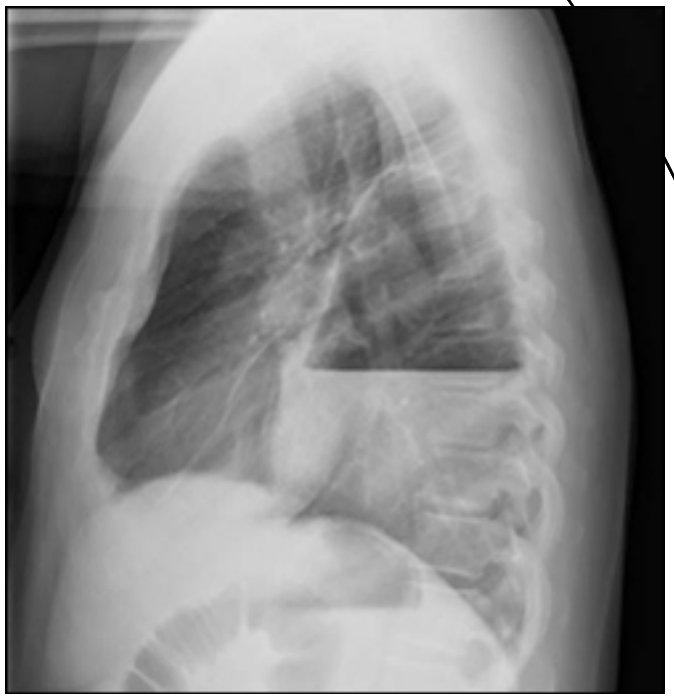
Extramedullary plasmacytoma

Plasmablastic lymphoma

Multiple myeloma

- **Extramedullary plasmacytoma**—This rare plasma-cell tumor originates as a soft-tissue lesion, without osseous or systemic involvement. Diagnosis requires a histologically confirmed single lesion showing monoclonal plasma cells and the exclusion of multiple myeloma.[1] The mainstay of treatment is tumoricidal radiation at a dosage of 40 to 50 Gy over 4 to 5 weeks.[2] Adjuvant chemotherapy has no proven benefit. However, thalidomide (Thalomid) has been used as palliative therapy in cases that are refractory to standard treatment or that have progressed to multiple myeloma.[2]
- Extramedullary plasmacytoma

- A 57-year-old black man with a history of type 2 diabetes, hypertension, dyslipidemia, and hepatitis C, presented to the emergency department with a 1-month history of right-sided chest pain associated with fever, cough, shortness of breath, and nausea.
- Physical examination revealed a temperature of 101°F and decreased breath sounds on the right side. Complete blood cell count with differential revealed: leukocyte count, 17.2 x 10^9/L (normal, 4.0-11.0 x 10^9/L), without neutrophilia; hemoglobin, 11.2 g/dL (normal, 13.5-17.5 g/dL); hematocrit, 33.6% (normal, 42%-52%); platelet count, 483 x 10^9/L (normal, 140-400 x 10^9/L). The initial routine chest radiographs revealed the diagnosis

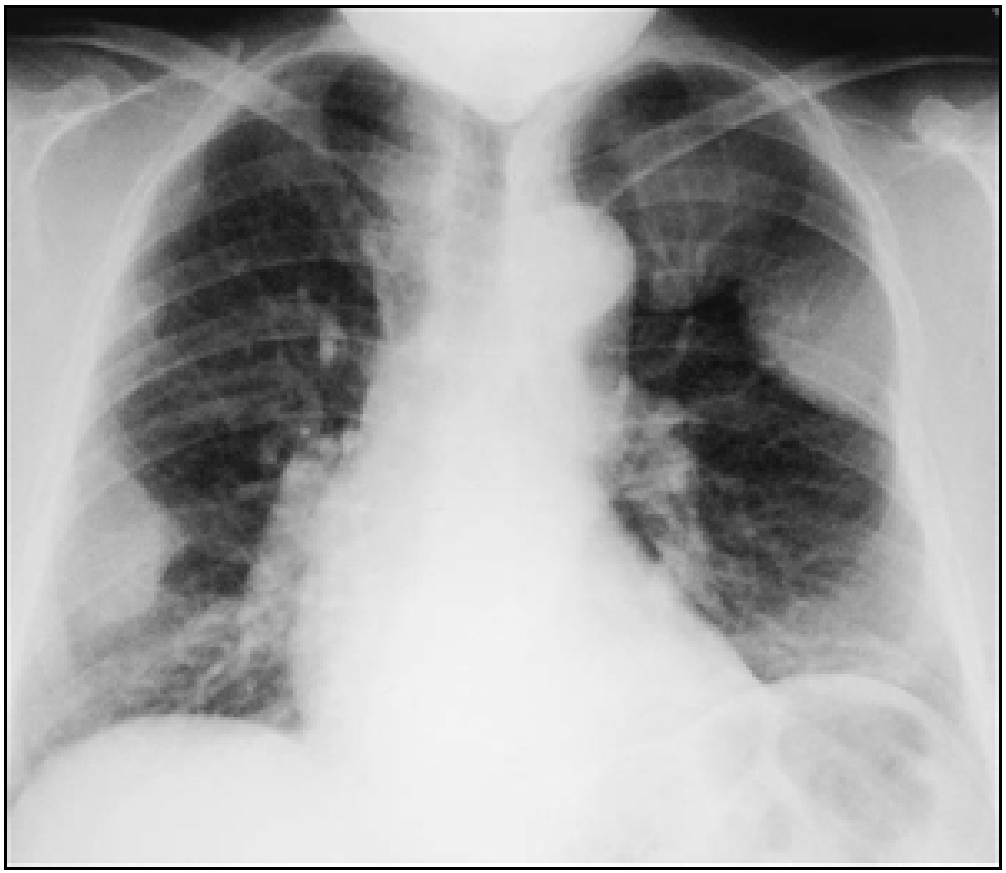


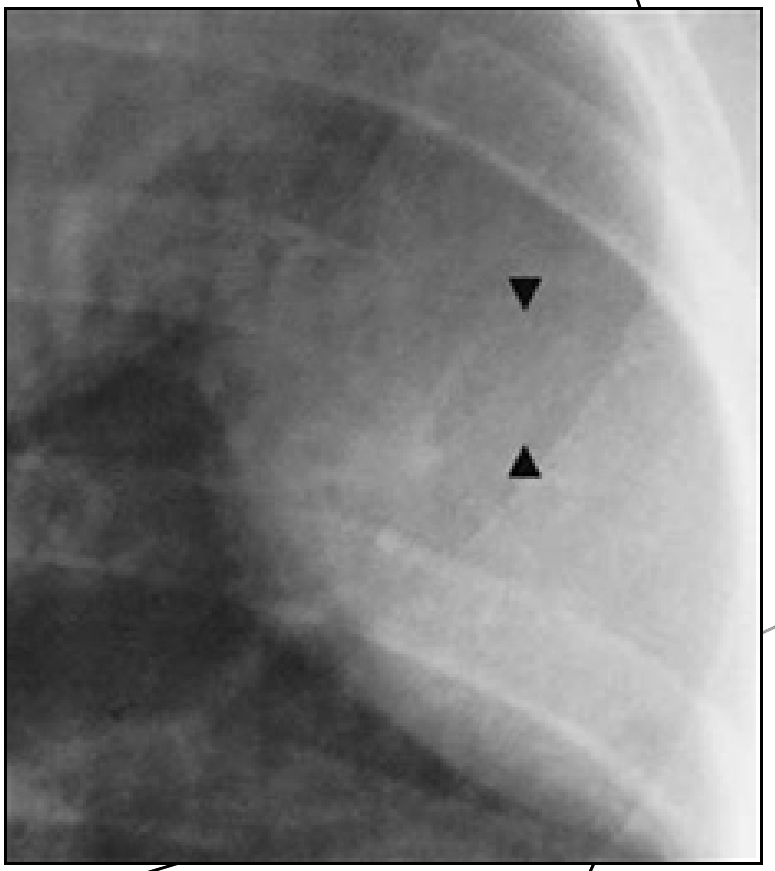
What's Your Diagnosis?

Lung abscess
Empyema
Hiatal hernia
Bronchogenic cyst

- **Empyema**-Our patient's symptoms and signs, as well as chest radiograph findings provided the clues to the diagnosis. A huge locule of air and fluid with an air-fluid level was seen in the right hemithorax (Figure). A subsequent computed tomography (CT) scan of the chest also showed a large fluid collection in the posterior right hemithorax consistent with a large empyema. The pleural fluid was cloudy green. Pleural fluid analysis showed: white blood cell count, 160 x 10^9/L (an accurate differential could not be performed because of the degenerated cells in an extremely purulent specimen); pH, 6.03; protein, 2.5 g/dL; lactate dehydrogenase (LDH), >2500 U/L; glucose, 274 mg/dL. The corresponding serum chemistries were: protein, 5.7 g/dL (normal, 6.6-8.2 g/dL); LDH, 296 U/L (normal, 90-220 U/L); glucose, 190 mg/dL (normal, 75-120 mg/dL). These findings were consistent with the diagnosis of empyema. Pleural fluid culture grew *Hemophilus influenza.* After a chest tube placement, the patient was given intravenous antibiotics, with subsequent clinical improvement

A 68-year-old man presented with pleurisy and cough of 2 weeks' duration. Physical examination, complete blood cell count, and urinalysis were all normal. A chest radiograph showed 3 large masses (Figure 1), each contiguous with a partially destroyed rib (Figure 2). Lytic lesions were also evident in the calvarium and left femur. The serum calcium level was elevated at 13.5 mg/dL (normal, 8.4-10.2 mg/dL), and serum electrophoresis showed a monoclonal spike of 4.4 g/dL. Biopsy specimens from bone marrow and one of the rib masses showed sheets of plasma cells, confirming the diagnosis of multiple myeloma





- **Points to remember:** The combination of hypercalcemia, lytic bone lesions, and a high monoclonal spike strongly suggests multiple myeloma. Definitive diagnosis, however, requires bone marrow examination or biopsy of a suspicious bone lesion.
- Chest disease associated with multiple myeloma can manifest as infection or as bone lesions, complicated at times by pleural effusion. A bone lesion may expand and appear radiographically as a "lung mass."
- **Diagnosis:** Multiple myeloma

A 45-year-old man with a history of chronic alcohol abuse, pancreatitis, and chronic, untreated sinusitis presented to the emergency department with complaints of abdominal pain and intermittent right-sided throbbing headaches that were nonradiating. Physical examination revealed a 3- x 3-cm nonpulsatile, nontender, fluctuant swelling over the right brow. The patient was admitted for further evaluation. Twenty-four hours postadmission, right-eye ptosis was evident, accompanied by an increase in the size of the swelling to 4 x 5 cm (Figure 1).

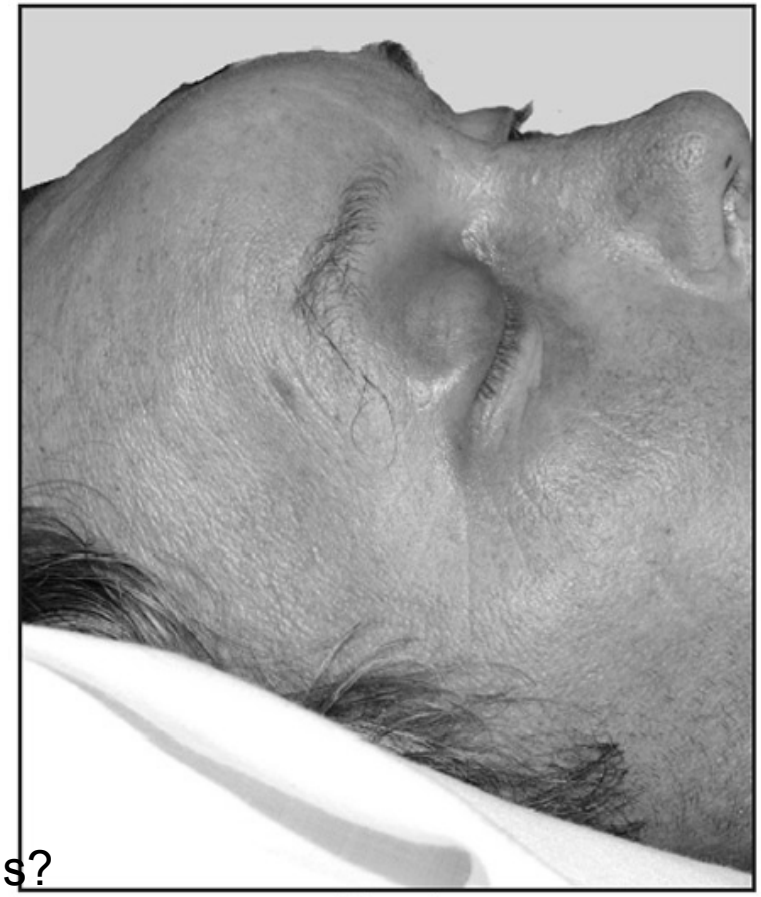


Figure 1

What's Your Diagnosis?

Inflamed epidermoid cyst

Cellulitis

Frontal bone osteomyelitis

~~Eosinophilic granuloma~~

- **Frontal bone osteomyelitis**—Also known as Pott's puffy tumor, this condition is defined as a subperiosteal abscess of the frontal bone associated with osteomyelitis. The introduction of antibiotics has rendered this condition a rare entity, most often presenting as a soft-tissue swelling overlying frontal bone osteomyelitis.
- The 2 major confirmed causes are chronic sinusitis and trauma, presumably occurring in the presence of frontal sinus infection or bacterial colonization.[1] The close proximity of the frontal sinus to the marrow cavity of the frontal bone makes the frontal sinus the most common sinus involved in osteomyelitis secondary to sinusitis. The infection can involve the inner and/or the outer table of the frontal bone. Thrombophlebitis in the valveless diploic veins of Breschet contributes to intracranial spread of the infection, because these veins provide a communication between the sinus mucosa and the bony trabeculae. Erosion through the anterior table of the frontal bone can lead to the formation of a subperiosteal abscess or to Pott's puffy tumor.[1,2]

A 25-year-old Nepalese woman presented to a primary care clinic complaining of right-sided neck pain, swelling, and fever for the past 3 weeks. She had no known allergies and denied any chills, cough, night sweats, and weight loss. Physical examination revealed a 3-cm tender, palpable mass below the angle of the jaw, along with palpable nodes in the inferior and posterior cervical chains. She was afebrile. Complete blood cell count showed: leukocyte count, 3.1 x 10^9/L, with 14% neutrophils; bands, 14%; lymphocytes, 41%; monocytes, 19%. Tests for viral infection were negative. Magnetic resonance imaging of the neck (Figure 1) and right cervical lymph node excisional biopsy for definitive diagnosis (Figures 2, 3) were performed

Figure 1

What's Your Diagnosis?
Sarcoidosis
Kikuchi's disease
Hodgkin's lymphoma
Castleman's dise

- **Kikuchi's disease**—Also known as histiocytic necrotizing lymphadenopathy, Kikuchi's disease is a rare, benign disorder of the lymph nodes in young adults. Although uncommon in Western countries, this disorder should be considered in the differential diagnosis of lymph node enlargement, because its course and treatment differ markedly from that of other causes. The exact etiology is unknown; viral or postviral etiology has been proposed.
- Kikuchi's disease presents with cervical lymphadenopathy accompanied by fever, myalgia, or neutropenia.[1,2] Other symptoms include night sweats, headache, malaise, fatigue, and arthralgia. On palpation, these nodes are tender, firm, and mobile, and without any drainage or fluctuation

- A 30-year-old (recent immigrant) Hispanic man presented to the emergency department with a 3-month history of left-sided pleuritic chest pain and difficulty breathing. The chest pain was accompanied by a 10-lb weight loss over 6 weeks, fever, and weakness, but no cough. A purified protein derivative (PPD) test performed during a recent incarceration was negative.
- Physical examination revealed he had mild tachycardia and decreased breath sounds in the left lung base. Sputum stain was negative for acid-fast bacilli or other bacteria. Chest radiograph revealed left lower lobe atelectasis with a large pleural effusion (Figure 1). Computed tomography of the thorax showed similar findings, with nodular thickening of the visceral and parietal pleurae (Figure 2).
- Empirical antibiotic treatment for community-acquired pneumonia was not helpful. A pleurocentesis done at admission yielded thick, lymphocytic, turbid, inflammatory fluid that clotted while awaiting laboratory findings. The microbiologic specimen was reported as “positive with acid-fast bacteria/high-power field.”

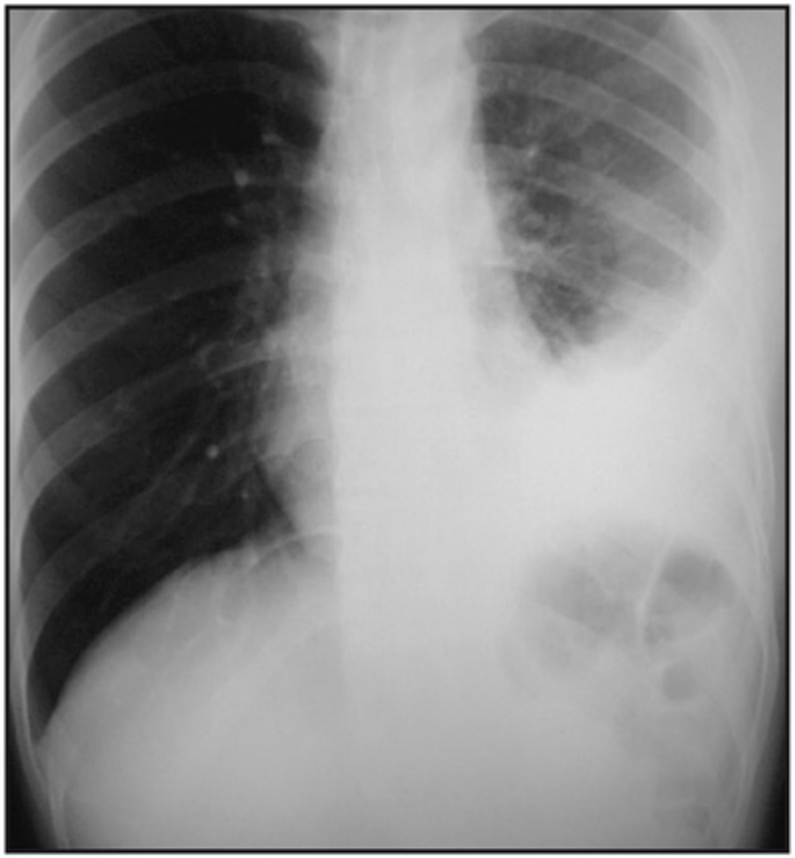


Figure 1

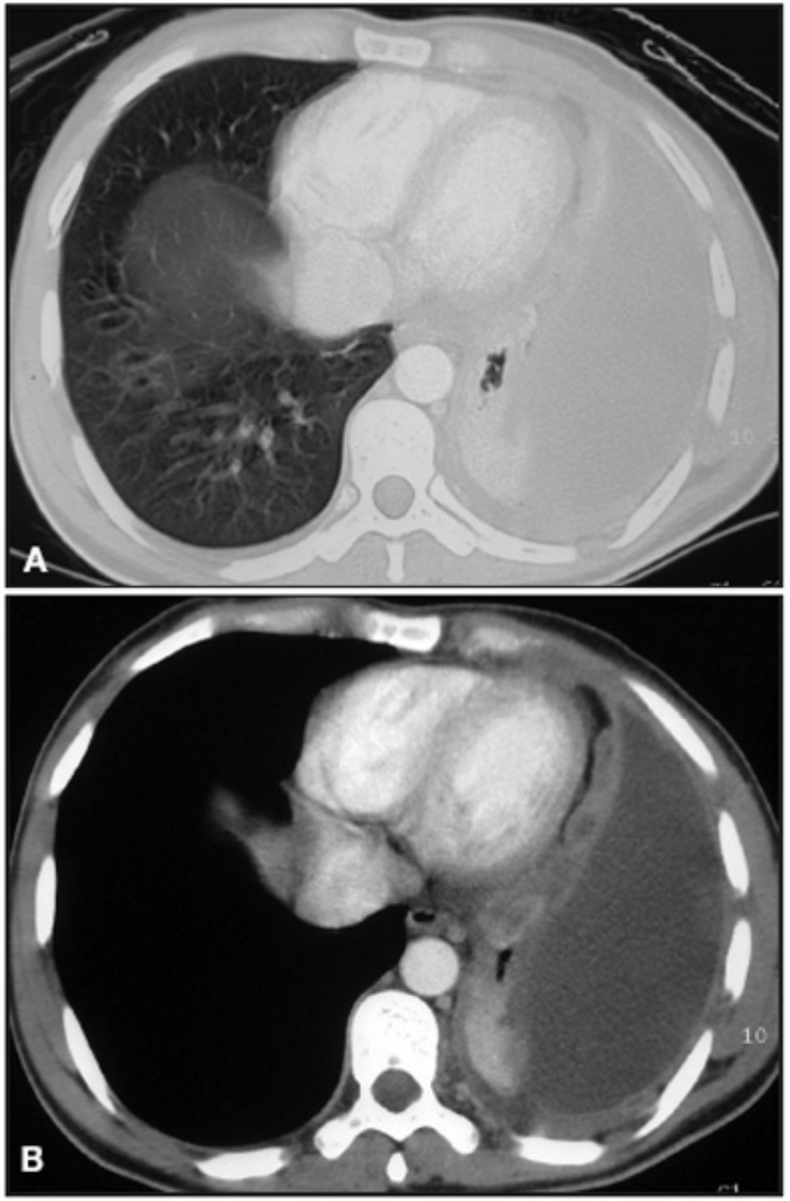


Figure 2

What's Your Diagnosis?
Pleural tuberculosis (TB)
Bacterial empyema
TB empyema
Actinomycosis empyema

- **TB empyema**—Based on the microbiologic findings, the patient was diagnosed with TB empyema and was prescribed rifampin, ethambutol, and isoniazid. His condition quickly improved, and he was discharged with prescriptions to continue the medications.
- The pleural cavity is one of the more common sites for extrapulmonary TB. Infection with TB of the pleural space can manifest as pleural effusion or empyema. The diagnosis of TB empyema differs from pleural TB by the presence of pus. In addition, the mycobacterial organism is found in large amounts on staining, as was seen in this patient.
- A negative PPD test may be present in acute or long-term TB infections. The standard anti-TB antibiotics may be used for TB empyema. The treatment may, however, need to be supplemented with surgical drainage, because of the limited penetration of the drugs into the pleural cavity.

A 57-year-old woman came to the emergency department with a complaint of precordial chest pain. Physical examination revealed: heart rate, 106 beats/min; blood pressure, 160/100 mm Hg; temperature, 99.7 °F; respiratory rate, 16 breaths/min. Her medical history included hypertension, diabetes, and coronary artery disease (CAD). A stent was placed in her right coronary artery 2 months before presentation. Cardiac examination showed no evidence of jugular venous distention, carotid bruit, or rate or rhythm abnormalities; she had normal S_1 and S_2, without any evidence of murmur. Both lungs were clear to auscultation. Abdominal examination was normal, and the lower extremities had no edema.

Figure 1

- **Diagnosis:** Coronary vasospasm with preexisting CAD.
- The ECG shows ST-segment elevation in the anterior leads. The patient was immediately taken to the cardiac catheterization laboratory with the suspected diagnosis of ST-segment elevation myocardial infarction. Coronary angiogram showed no significant disease in the left main coronary artery, which trifurcated into the left anterior descending artery, the left circumflex artery, and a ramus intermedius coronary artery. The left anterior descending artery had mild luminal irregularities. The left circumflex artery had a 50% lesion in the distal portion. The right coronary artery had a patent stent in the midportion of the vessel. No coronary intervention was performed in the cardiac catheterization laboratory, and the patient was transferred to the coronary care unit for medical management. The patient was free of chest pain at that time. Repeated ECG showed resolution of the ST-segment elevation in the anterior leads (Figure 2). Coronary vasospasm, with preexisting CAD, was diagnosed, and nitrate treatment was started.

An 84-year-old man with Crohn's disease presented to the emergency department with a rigid, distended abdomen associated with obtundation and respiratory failure requiring mechanical ventilation

Figure 1

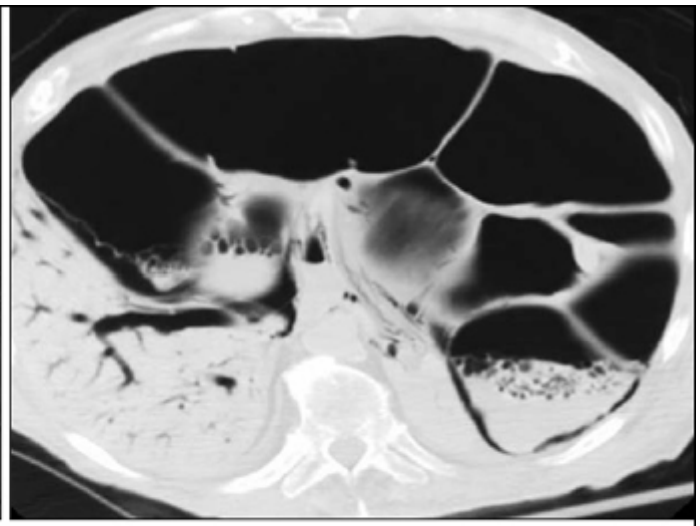


Figure 2

What's Your Diagnosis?
Colonic perforation

Small bowel obstruction
Colonic volvulus with mesenteric ischemia

~~Ogilvie's syndrome~~

- **Colonic volvulus with mesenteric ischemia**—This diagnosis must be considered in the differential diagnosis of the elderly patient presenting with abdominal pain and distention. This patient's CT images reveal classic findings associated with mesenteric ischemia: marked distention of the small and large bowels, with pneumatosis intestinalis and extensive portal venous gas

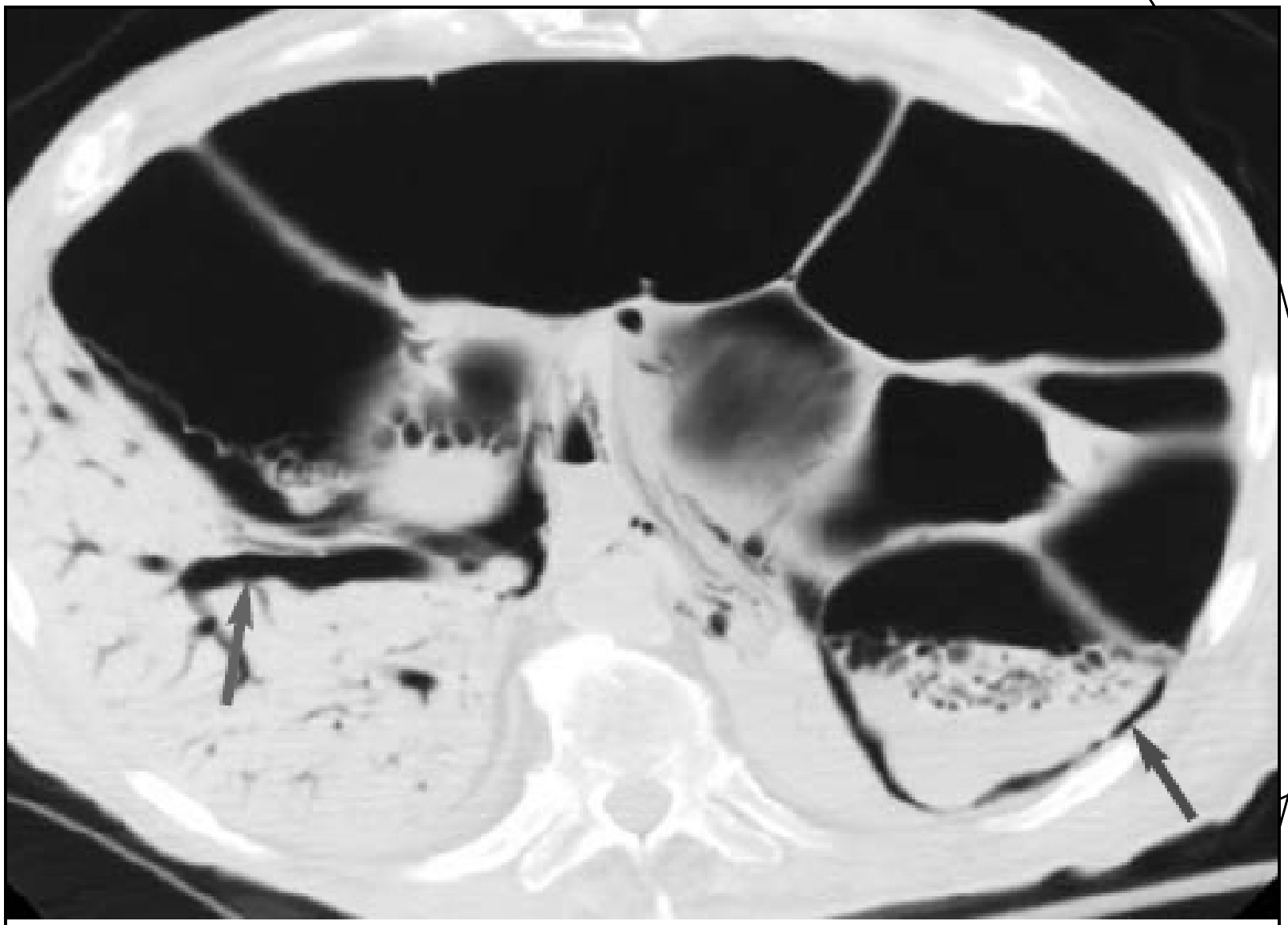


Figure 2 - Pneumatosis intestinalis (red arrow) and diffuse portal venous gas (blue arrow) characteristic of mesenteric ischemia.

- A 65-year-old woman presented with abdominal pain. Physical examination showed abdominal distension, ascites, and epigastric tenderness. Serum lipase value was 2594 U/L and ionized calcium concentration was 0.5 mg/dL. Two days after admission, periumbilical bruising appeared (Figure). Computed tomography scan of the abdomen revealed extensive pancreatic edema without evidence of hemorrhage. After institution of hypertriglyceridemia therapy and 7 weeks of supportive care, the pancreatitis resolved. The periumbilical bruising lasted for 2 weeks

- Ecchymotic discoloration of the abdominal wall near the midline, from the umbilicus to the symphysis pubis, is known as Cullen's sign. It results from retroperitoneal or intraperitoneal bleeding, with blood reaching the abdominal wall by way of the falciform and round ligaments. First described by Thomas Cullen in 1918 in association with ectopic pregnancy, this sign occurs only in 1% to 2% of patients with acute pancreatitis.
- Points to remember: In the absence of trauma or blood disorders, the appearance of Cullen's sign is a valuable bedside clue to retroperitoneal or intraperitoneal hemorrhage.

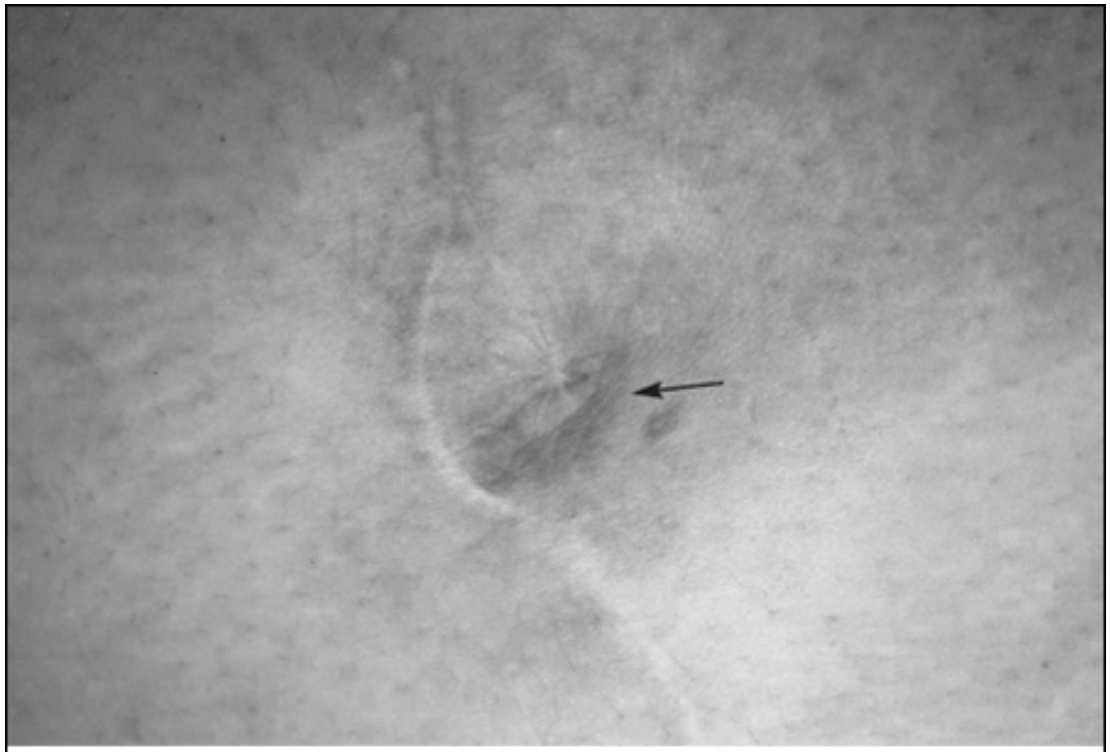


Figure - "Purplish" periumbilical ecchymosis (arrow).

Diagnosis: Acute pancreatitis with intraperitoneal hemorrhage.

A 90-year-old nursing home resident with Alzheimer's dementia was found to have extensive rashes on the soles of her feet (Figure) and a few papular lesions on her palms. She denied itching or pain. She had no other systemic signs or symptoms and no other lesions. Her medical history included a positive rapid plasma reagin (RPR) test done 9 months ago.

What's Your Diagnosis?
Syphilitic lesions
Pustular psoriasis
Chronic eczema
Dermatitis herpetiformis

- **Localized pustular psoriasis**—This type of psoriasis, normally found on the palms of the hands and/or the soles of the feet, looks very different from plaque or flexural psoriasis. Unlike plaque psoriasis, red, scaly lesions are not the predominant feature of pustular psoriasis. Instead, it has a mass of weeping, cracked areas that look like small, yellowish blisters. These can be very painful and sore and seem to be infected, although no infection is present. The pustules can turn brown over a matter of days and fall off, only to be replaced by new yellow blisters. This turnaround can occur many times and may last for years. In this patient, a 4-mm punch biopsy of a sole lesion was suggestive of psoriasis. Application of a topical corticosteroid resulted in improvement of the lesion.
- Secondary syphilis is characterized by a rash that is classically a symmetric papular eruption on the entire trunk and upper and lower extremities. Large, raised, gray-to-white lesions (condyloma lata) involving warm, moist areas (eg, mucous membranes in the mouth and perineum) may develop in some patients. Our patient had no other systemic symptoms or lesions. The only positive result was a low RPR titer (1:1).
- Chronic eczema involves areas of red, itchy skin. This condition usually starts in early childhood, especially with a family history of atopy (asthma, hay fever, conjunctivitis, or food allergies).
- Dermatitis herpetiformis is an autoimmune blistering disorder associated with a gluten-sensitive enteropathy. It is characterized by grouped excoriations; erythematous, urticarial plaques; and papules with vesicles. These lesions are located on the extensor surfaces of the elbows, knees, buttocks, and back but do not affect the palms and soles. Direct immunofluorescence of a skin biopsy characteristically shows deposition of immunoglobulin A in a granular pattern in the upper papillary dermis, unlike psoriasis.
-

A 52-year-old woman with acute myelogenous leukemia presents with a 2-day history of 102°F fever, right lower-quadrant abdominal pain with distention, nausea, vomiting, and watery diarrhea. She had received high-dose chemotherapy 10 days ago. Workup shows: hemoglobin, 8.4 g/dL; absolute neutrophil count, 50/µL; platelet count, 25 x 10^3/µL. Computed tomography (CT) of the abdomen and pelvis confirmed the diagnosis (Figure).

What's Your Diagnosis?

Acute appendicitis

Typhlitis

Pancreatitis

Diverticulitis

- **Typhlitis**—This necrotizing enterocolitis occurs mainly in patients with immunosuppression, hematologic malignancies, neutropenia, or after cytotoxic chemotherapy. Typhlitis results from a combination of impaired host defense to invasion by microorganisms, mucosal injury, and profound neutropenia. The infection leads to necrosis of various layers of the bowel wall. The cecum is almost always affected, and often extends into the ascending colon and the terminal ileum. The predilection for the cecum is related to its diminished vascularization relative to the rest of the colon. Gross and histologic examinations may reveal bowel-wall thickening, discrete or confluent ulcers, mucosal loss, intramural edema, hemorrhage, and necrosis.[1]
- Typhlitis should be considered in any neutropenic patient who presents with fever and right lower-quadrant abdominal pain. Symptoms may include abdominal distension, nausea, vomiting, and watery or bloody diarrhea. Stomatitis and pharyngitis, suggesting the presence of widespread mucositis, are usually present. Symptoms usually appear 10 to 14 days after chemotherapy, when neutropenia is most profound.[2] Peritonitis, perforation, and severe bleeding are possible complications

- A 46-year-old man presented with increasingly yellow skin discoloration evident on his face (Figure 1), torso, and extremities but not on the mucous membranes or sclerae. Symmetrical palm and sole erythemawith desquamation at the pressure points was also noted (Figure 2).
- Twelve months ago, the patient had been diagnosed with kidney cancer, which had metastasized to his lungs, liver, and bones. He was prescribed oral therapy with one of the multitargeted tyrosine kinase inhibitors 1 week ago.
- The patient denied taking any vitamin supplements, any changes in his urine or stool, itching or fever, or consuming excessive amounts of carotene-containing juices or foods. All laboratory test results were normal

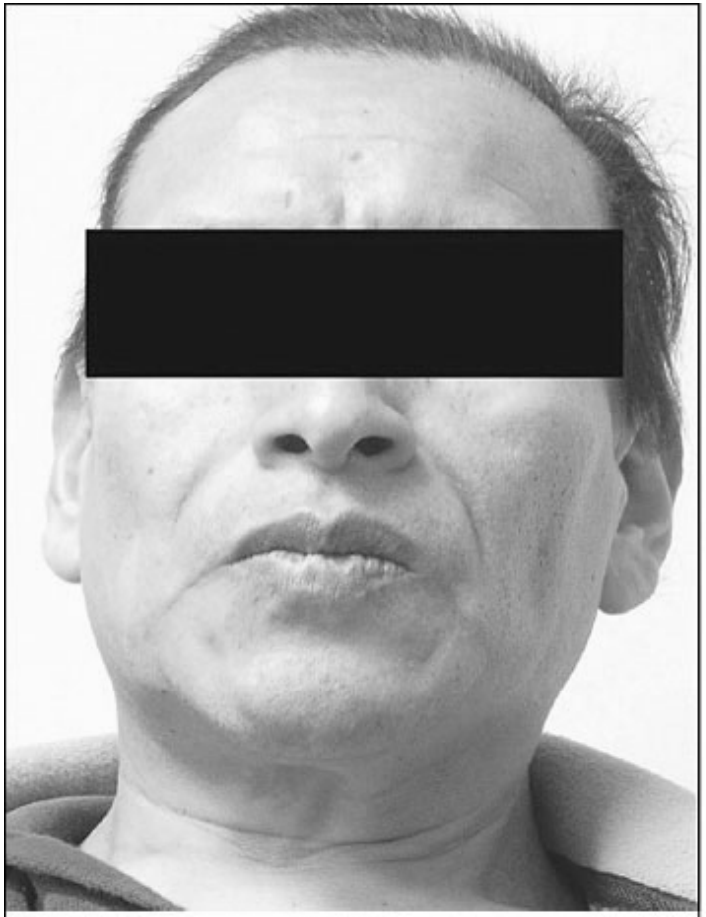


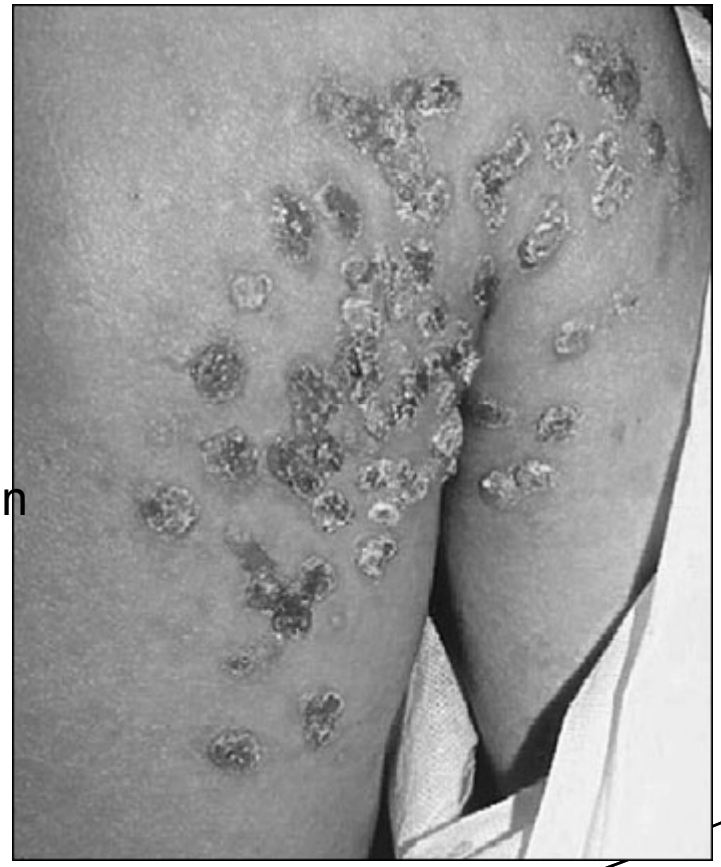
Figure 1

Figure 2

What's Your Diagnosis?
Jaundice
Medication side effect
Cobalamin deficiency
Carotenemia

- **Medication side effec—**The yellow skin discoloration seen in this patient represents a side effect associated with the newly approved multitargeted tyrosine kinase inhibitors, sorafenib (Nexavar) and sunitinib malate (Sutent), which are used to treat renal-cell carcinoma. Two key findings support this diagnosis: the occurrence of the skin discoloration within 1 week of starting sorafenib therapy, and the presence of hand–foot syndrome.[1]
- The yellow pigmentation associated with tyrosine kinase inhibitor therapy spares the sclerae and mucous membranes and is especially pronounced on the palms and soles. Discoloration increases at high doses and with continued treatment, suggesting that the effects may be related to the excretion of these drugs and/or their metabolites through the skin.[1] This aesthetically unpleasant side effect disappears within weeks of drug discontinuation. A superimposed hand–foot reaction is seen in nearly 20% to 30% of patients taking these agents.[1,2]Although its severity varies, the hand–foot syndrome typically manifests as small blisters on the palms and soles that heal with peeling.

A 25-year-old Japanese man returning to Japan from a vacation in Jamaica was transferred from a local airport (during a stopover in the United States) for evaluation of a suspicious neck lesion that was noticed by a fellow passenger. His medical history revealed no constitutional symptoms or animal contacts, but he reported that he had had 2 sexual encounters while in Jamaica. Physical examination revealed multiple circular vegetative, erythematous pustules with thick yellow crusts on the chest, abdomen, axilla, lower extremities, and upper back (Figure).



What's Your Diagnosis?

Allergic contact dermatitis

Pediculosis

Nonbullous impetigo

Herpes simplex virus infection

- **Nonbullous impetigo—**This contagious, superficial bacterial infection is caused by *Staphylococcus aureus*, group A streptococcus (*Streptococcus pyogenes*), or a com-bination of both. In this common skin infection, lesions develop at the site of a minor trauma when the integrity of the skin is disrupted, thereby allowing the organisms to enter the epidermis. After excoriating an infected area, patients can spread the infection through physical contact. This mode of transmission primarily affects the pediatric population, as the microorganism can easily be disseminated at day care centers, nurseries, or grade schools. Adults usually contract the infection from children, or when their personal hygiene is poor, when they live in crowded conditions, or work in an unhygienic environment

- A 16-year-old Hispanic male presented to the emergency department with a 3-day history of difficulty walking because of pain in the right groin, discomfort in the posterior aspect of the left thigh, and fever up to 104°F. The symptoms were sudden in onset. The patient denied local trauma or intravenous drug use. Physical examination findings included temperature, 102°F; pulse, 136 beats/min. A 10-cm cigar-shaped, firm, tender subcutaneous mass was evident perpendicular and inferior to the right inguinal ligament; the skin was warm and slightly erythematous. A 4- to 5-cm area on the back of the left thigh was tender. Muscle strength and passive range of motion was normal throughout. Laboratory test results included: white blood cell count, 20.4 x 10^6/μL, with 93% neutrophils; erythrocyte sedimentation rate, 48 mm/h; creatine phosphokinase level, 456 U/L. Computed tomography (CT) (Figure 1) and magnetic resonance imaging (MRI) (Figure 2) scans are shown.

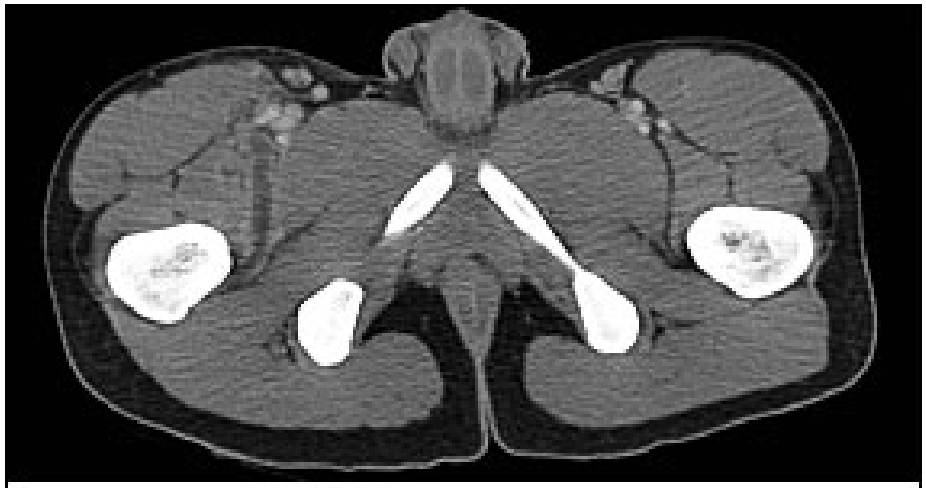


Figure 1

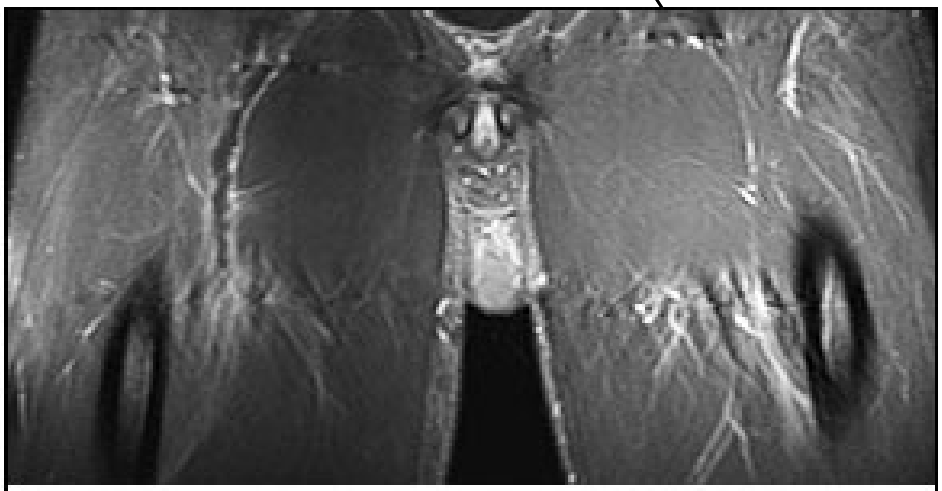


Figure 2

What's Your Diagnosis?

Polymyositis
Pyomyositis
Dermatomyositis
Inclusion body myositis

- **Pyomyositis**—This patient is only the third reported case of pyomyositis caused by infection with methicillin-resistant *Staphylococcus aureus* (MRSA) in an otherwise healthy individual. The other 2 cases were mentioned in a letter published last year.[1] Pyomyositis itself is rare in the United States[2] and usually occurs in immunosuppressed patients, especially those with AIDS.[3] It is more common in tropical areas and is seen in children and in the malnourished population.
- Infection

An 80-year-old woman was admitted to the internal medicine service after a mechanical fall at home. She had a history of chronic low back pain. Her functional status had been declining at home despite maximal medical therapy for pain.
Magnetic resonance imaging (MRI) (with fat saturation) of her lumbar spine was performed. A sagittal T2-weighted MRI scan (Figure 1) and a coronal T1-weighted postgadolinium MRI scan (Figure 2) are shown.

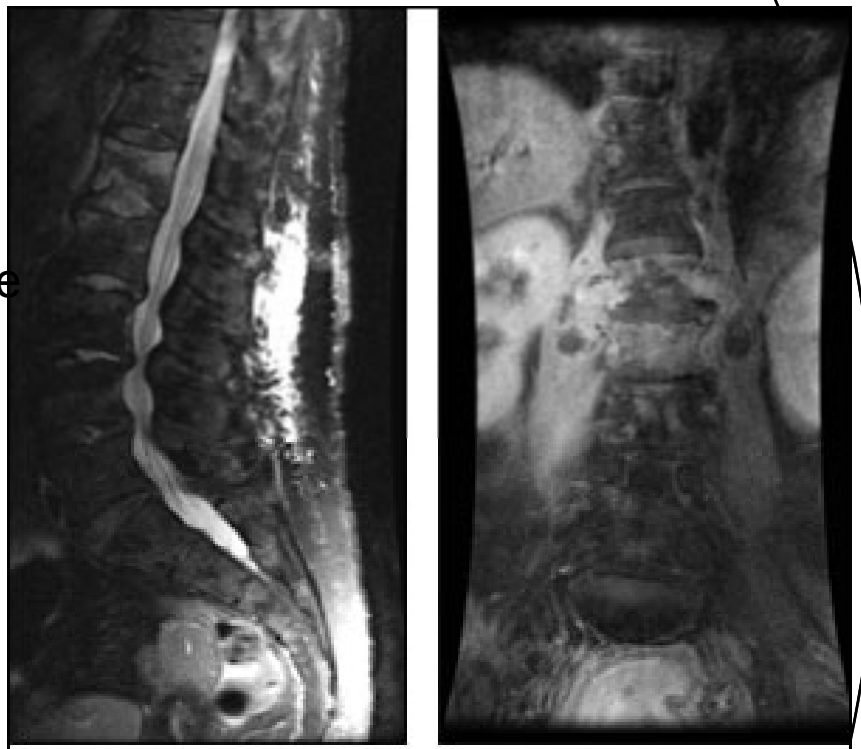


Figure 1 Figure 2

What's Your Diagnosis?

Osteoporosis with compression fracture

Metastatic disease

Spondylitis with paraspinal abscess

Hemodialysis-related arthropathy

- **Spondylitis with paraspinal abscess**—The sagittal T2-weighted MRI demonstrated a compression deformity of vertebra L1, high T2 signal involving vertebrae L1 and L2, disk herniation into the inferior endplate of L1, and spondylotic changes from L2/L3 through L5/S1. The coronal T1-weighted postgadolinium MRI demonstrated enhancement of vertebrae L1 and L2. Bilateral enhancement within the psoas muscles was seen adjacent to the L1 and L2 vertebrae, with small intramuscular peripherally enhancing fluid collections; these findings were suggestive of abscesses.
- Computed tomography (CT)–guided fine-needle aspiration of one of the abscesses was performed. Initial tests were negative, but purification and 2-week culture of the aspirate produced acid-fast bacilli, which were identified as *Mycobacterium tuberculosis.* The patient was started on a 9-month 3-drug regimen for tuberculosis (TB) and was discharged to a nursing facility.
- Although TB spondylitis (Pott's disease) is no longer common in developed countries, it remains common in developing countries.[1,2]

A 55-year-old obese black woman presented with extensive, painful, necrotic skin lesions on her anterior abdominal wall, back, and upper thighs (Figure 1). Her medical history included hypertension and end-stage renal disease, for which she received daily peritoneal dialysis. She denied any antecedent trauma or use of anticoagulants. Laboratory test results were: serum calcium, 1.98 mmol/L; phosphorus, 4.1 mmol/L; serum albumin, 16 g/L; parathyroid hormone, 410.78 pmol/L; white blood cell count, 0.021 x 10^9/L. A skeletal survey showed generalized demineralization affecting multiple bones. Skin biopsy was performed (Figure 2). Despite aggressive and appropriate therapy, the patient died from sepsis complications.

Figure 1

What's the Diagnosis?
Warfarin-induced skin necrosis
Pyoderma gangrenosum
Calcific uremic arteriolopathy
Lupus panniculitis

- **Calcific uremic arteriolopathy**—Also known as calciphylaxis, this condition is typically seen in patients receiving renal replacement therapy, with an estimated incidence of 1%.[1]Predisposing factors to calciphylaxis (among patients receiving renal replacement therapy) include obesity, female gender, white race, diabetes mellitus, calcium supplementation, warfarin therapy, and hypoalbuminemia. Metabolic derangements include hypercalcemia, hyperphosphatemia, secondary hyperparathyroidism, and an elevated calcium-phosphate product. This disorder consists of small-vessel calcification and necrosis of subcutaneous fat secondary to reduced perfusion and vascular thrombosis. Biopsy is useful for confirmation. Figure 2 shows extensive calcification involving the intima and lumen of a medium-sized artery. Intimal hypertrophy, medial calcification, and thrombosis of the vessel lumen are all seen in calciphylaxis

A healthy 24-year-old male physician had worked in a local clinic in Senegal and Cote d'Ivoire for 2 weeks. During his stay, he visited a beach, where he went swimming and played volleyball. Six days later he noticed several raised, red, extremely pruritic lesions on his right foot

What's the Diagnosis?
Scabies
Cutaneous leishmaniasis
Cutaneous larva migrans
Bacterial cellulitis

Figure

- **Cutaneous larva migrans** (creeping eruption)—This is among the most frequent skin conditions acquired by travelers to the tropics.[1] It affects visitors to Africa, Latin America, the Caribbean, Southeast Asia, and the southern United States. Cutaneous larva migrans is caused by infection from hookworm larvae found in soil contaminated with animal feces. Most cases are due to hookworm species that infect dogs and cats; humans are incidental hosts. Adult worms in the animal host's gut produce eggs that are shed in fecal matter and develop into infectious larvae in warm, moist soil. Travelers usually become infected by sitting or walking barefoot on beaches or other soil contaminated with animal feces.

A 76-year-old retired physician came to the clinic for a medical check-up. He had never experienced any serious medical problem and had no history of heart disease. His physical examination was unremarkable. A chest x-ray was ordered, revealing prominence of the right heart border in the area of the ascending aorta, which was not seen on an old x-ray taken 7 years earlier. His electrocardiogram (ECG) revealed some irregularities (Figure 1).

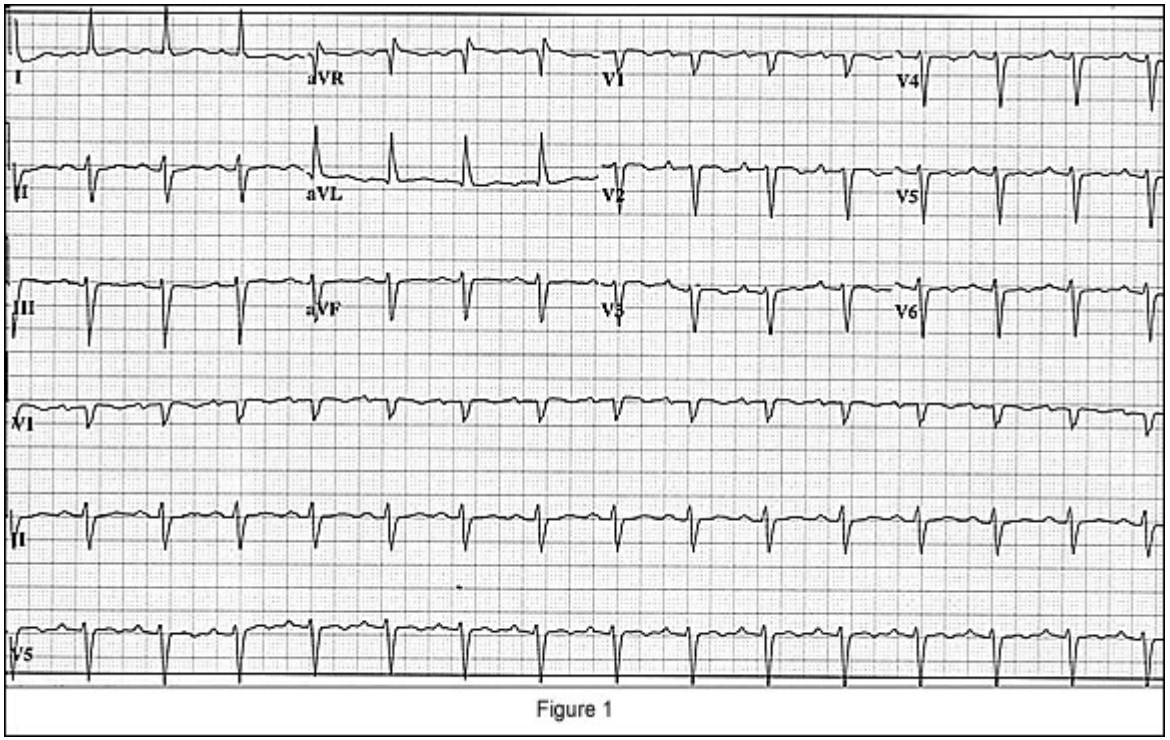


Figure 1

Questions: **Does the ECG show any specific heart disease? How do you explain the loss of anterolateral R-wave forces? Is it a "Q-wave equivalent" and a marker of previous silent myocardial infarction (MI) in this patient?**

- Diagnosis: **Left anterior fascicular block.** The characteristics of left anterior fascicular block include leftward QRS axis of –45° or more and evidence that this axis shift is not the result of an inferior wall MI. The diagnosis of a conduction block is considered when the QRS duration is prolonged. In such a case of left anterior fascicular block, the QRS duration remains normal, since only 1 fascicle is involved, which does not cause a significant increase in the QRS duration. The evidence for no inferior MI is fulfilled by the pattern of an insignificant Q wave in lead I and an R wave in lead III, which indicate that much of the septal depolarization is normal.

A 36-year-old woman presented with general malaise, fever, and fatigue. Physical examination showed marked bilateral cervical lymphadenopathy, which was slightly more prominent on the right side. A complete blood cell count revealed leukocytosis, with mild anemia. A superficial fine-needle aspiration of a lymph node on the right side was performed (Figure 1), and a biopsy was later obtained (Figure 2), confirming the diagnosis.

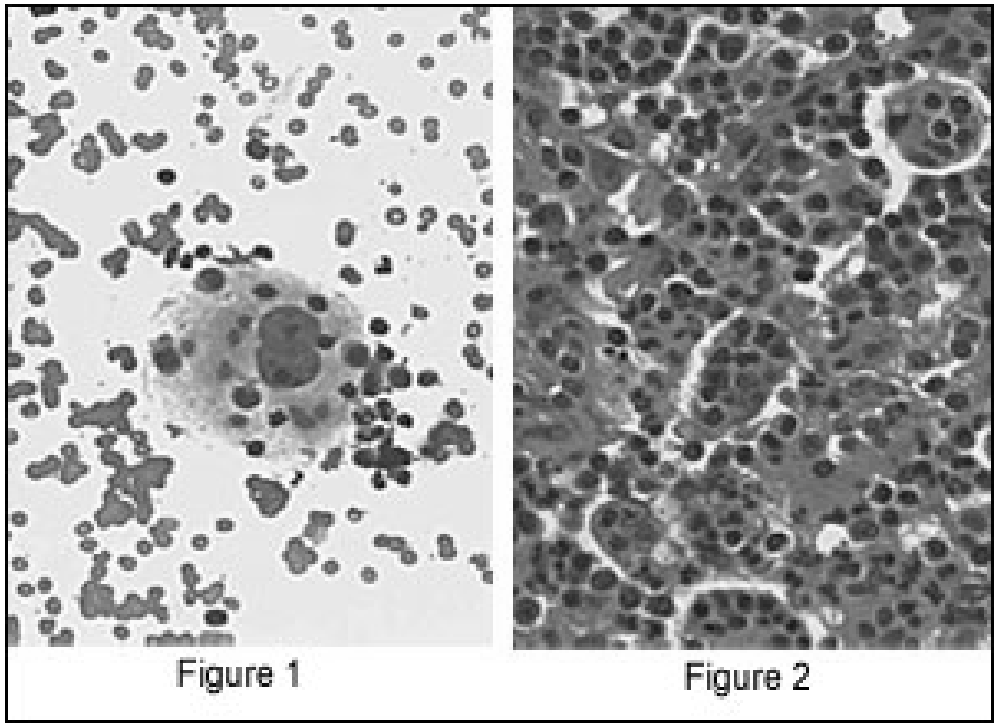


Figure 1 Figure 2

What's Your Diagnosis?

Hodgkin's lymphoma

Rosai-Dorfman disease

Non-Hodgkin's lymphoma

Metastatic melanoma

- Answer: Rosai-Dorfman disease
- Also known as sinus histiocytosis with massive lymphadenopathy (SHML), this uncommon disorder was first described by Rosai and Dorfman in 1969. Clinically, the patient often presents with bilateral enlargement of the cervical lymph nodes, sometimes with fever and leukocytosis. The disease may also be present in extranodal sites. SHML often regresses spontaneously, but a minority of patients have a more aggressive course.
- The fine-needle aspiration shows a binucleate histiocyte containing lymphocytes and other white blood cells (Figure 1). The lymphocytes are traversing through the histiocyte's cytoplasm unharmed, a process known as emperipolesis. The binu-cleate nature of the histiocytes seen in some of the aspirated cells could potentially lead to diagnostic confusion with the binucleate Reed-Sternberg cells characteristic of Hodgkin's lymphoma. Although less likely, the atypical cells of SHML could also be mistaken for metastatic disease, particularly for malignant melanoma or non-Hodgkin's lymphoma.
- Emperipolesis, however, is pathognomonic for SHML and is, therefore, diagnostic of SHML. The only other human cells demonstrating emperipolesis are normal megakaryocytes. Normal histiocytes do not show emperipolesis. The biopsy specimen also shows numerous histiocytes packed with white blood cells (Figure 2). These histiocytes fill the lymph node sinuses, causing the sinus histiocytosis and the massive lymphadenopathy, as suggested by the name itself (ie, SHML